# SURGERY

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## Original Communications

### *Halsted Centenary*

### THE WILLIAM STEWART HALSTED CENTENARY

EMILE HOLMAN, M.D., SAN FRANCISCO, CALIF.

TO ALFRED BLALOCK we owe the inspired plan to celebrate, in 1952, the Centenary of Dr. Halsted's birth, and thereby honor America's most illustrious surgeon, whose precepts and practices are followed wherever on this planet modern surgery is performed. The Centenary was planned to be held in conjunction with the annual meeting of the Society of University Surgeons. Subsequently, the Society of Clinical Surgery and The Halsted Club were invited to participate in the celebration. A committee consisting of Emile Holman, chairman, Alfred Blalock, B. Noland Carter, George Finney, M. W. Firor, Mark Ravitch, W. F. Rienhoff, Jr., H. B. Shumacker, H. W. Scott, Jr., and Edward S. Stafford began, early in 1950, to make the necessary arrangements. In preparing the program, the Committee hoped to shed new light on the life of this shy, scholarly patrician, and to gain new information concerning his perplexing personality.

It seemed singularly appropriate to honor Dr. Halsted at this particular time as there were still a number of his intimate associates and devoted disciples who could be called upon to present personal recollections of their former chief. Among these were James Mitchell of Washington, Samuel Crowe of Baltimore, Willis Gatch of Indianapolis, Emil Goetsch of Brooklyn, Frederick Reichert of San Francisco, George Heuer of New York, and Roy McClure of Detroit. Before the year was over, death had claimed Dr. Heuer and Dr. McClure. Other intimate friends were called upon to present their recollections, including Edwards Park and Arthur Shipley of Baltimore. Miss Margaret Boise, his first full-time nurse anesthetist, kindly consented to speak. Dr. René Leriche of Paris, one of Dr. Halsted's most respected and devoted friends, was invited to speak at the Centennial Dinner as was also another great admirer of the Professor, Dr. Allen Whipple. The committee hoped fervently that Dr. Rudolph

Matas of New Orleans would be able to participate personally, but an almost complete blindness combined with other infirmities that accompany the age of 91 prevented his coming. His spirit, however, hovered ever near in appreciative homage to his dear friend. Indeed, his presentation to the Halsted Memorial Room of two volumes of personal letters from and to Dr. Halsted, beautifully and permanently bound, provided great pleasure to all who saw them on display in the exhibit of Halsted memorabilia. Other accessions to the exhibit of great interest were his gold watch and chain, a present from the former Mrs. Mont Reid, now Mrs. Albert Cash of Cincinnati, his study lamp from Dr. Crowe of Baltimore, his black top hat worn daily in the early days of the Hospital from Dr. Henry Peterson of Houston, his own stethoscope and the walking cane he habitually used from Emile Holman. It is hoped that former associates will generously contribute any similar memorabilia in their possession for permanent display in the Halsted Memorial Room now being built into the new surgical building.

At the Centenary exercises a beautiful bronze bust of Dr. Halsted executed by Dr. Vladimir Fortunato was presented to the Hospital by Dr. William F. Rienhoff, Jr.

Anticipated by many friends of Dr. Halsted and made available at the Centenary was the sympathetic account by Dr. Heuer of his long association with the Professor, published as a supplement to the Johns Hopkins Bulletin under the simple title "Dr. Halsted." It provides an admirable and intimate picture of the Professor in his relations with patients, students, surgical staff, and associates in the medical school.

Also made available at the Centenary by the Johns Hopkins Press were the two volumes of Dr. Halsted's complete contributions to surgery edited by Walter Burket—volumes which had been out of print for a number of years and which have been eagerly but vainly sought by recent generations of surgeons.

Invitations to attend the Centenary were sent to a number of foreign surgeons. The following responses from two illustrious surgeons will interest their many friends in this country:

Roma—Via dei Villini 2
January 19, 1952

The Halsted Centennial Committee
Emile Holman, M.D., Chairman
Dear Dr. Holman:

I have received your letter of January 12th in which you inform me that the Halsted Centennial Committee kindly invites me to attend the meeting which will take place at The Johns Hopkins Hospital February 7th. I thank you for this communication and I beg you to thank for me the members of the Committee.

This kind invitation is very much appreciated by me and I would feel happy if I could attend the meeting, which I am sorry to say is not possible.

I will spiritually join you and all the members of the Committee and of the other Societies, for I will think of my first acquaintance with Professor Halsted in 1905 and with his associates and assistants who with their work so highly honored their teacher and so deeply contributed to the progress of the science and art of surgery. What I could see and learn from him, from all his staff, and from the Johns Hopkins work in general was a great

lesson and inspiration for me which lasted through all my life. On February 7th the spirit of Professor Halsted will enjoy the greatest satisfaction ever attained by a teacher, to see his school standing higher and higher in the hands of the surgeons which have followed the master.

To them, too, I send my best wishes for future achievements in the science and art of surgery initiated at the Johns Hopkins by Professor Halsted.

Sincerely yours,
Raffaele Bastianelli

Uppsala,
January 28, 1952

Halsted Centennial Committee
The Johns Hopkins Hospital
Baltimore, Maryland
Dear Sirs:

I have very much appreciated your kind invitation to the celebration of the 100:d anniversary of the birth of Dr. William S. Halsted.

As K. G. Lennander's assistant in Uppsala, I visited Johns Hopkins Hospital in the spring of 1905, being, as I believe, the first Swedish surgeon crossing the Atlantic for the purpose of improving in surgical knowledge. At that time Europe did not pay much attention to American medical achievements.

My impressions of the new American surgery in its youthfully vigorous and bright evolution were really amazing and overwhelming. I remember Halsted in the operating room as the wise, skillful surgeon and taking part with all of us young men in our common lunch, and Kelly's remark to me: "You see we are all boys here." I remember Cushing carefully founding the new epoch of neurosurgery with painstaking operations in the surgical clinic and the Church Home and the delightful hospitality which he and his young wife gave me in their lovely home with their 20 months old baby as its center. And in my memory I keep a vivid impression of Young's splendid work in his new urological clinic. And all the other young surgeons and pathologists—Finney, Bloodgood, etc.—joined under the inspiring lead of Halsted and giving the young Swede generously all the best of their experiences!

But there were also other rich fountains of inspiration. Professor Welch's lectures in the history of medicine contributed to the atmosphere of fine culture. And not least the noble personality of William Osler! With all the members of the staff I participated in the grief of knowing him before long leaving Johns Hopkins for Oxford.

And so my attachment and admiration for American medicine got a foundation those days, which has become ever firmer during soon past 50 years.

I regret not to have the opportunity of being with you at the centenary and of reviving the old days in Johns Hopkins. And I ask you to give my regards to colleagues who, may be, still remember me.

Sincerely yours,
/s/Gunnar Nystrom
Professor Emeritus of Surgery

In retrospect, this first Centenary of Dr. Halsted's birth was a most satisfying and heart-warming experience. Most appropriately it was held in his own clinic, where the present surgical staff under the leadership of Dr. Alfred Blalock is making notable and epochal contributions to surgery. The operations performed, the papers presented, the recollections of Dr. Halsted by personal friends all contributed to make this Centenary one of the most memorable occasions in the history of American surgery. We may confidently predict that it marks only the first of many similar celebrations in centuries to come.

The program as presented included the following sessions and titles:

MEETINGS OF SOCIETY OF CLINICAL SURGERY AND THE HALSTED CLUB

WEDNESDAY, FEBRUARY 6, 1952

*Dr. C. Reid Edwards presiding*

The Treatment of Aneurysms With Polythene—Dr. R. Adams Cowley

The Henry Approach to the Repair of Femoral Hernia—Dr. Harry C. Hull

The Effect of "Cortone" in the Prevention of Intestinal Adhesions—Dr. William S. Lynn

A New Type of Pull-Out Sutures for Tendon Repair—Dr. Erwin R. Jennings

Intestinal Antibacterial Agents—Drs. E. R. Shipley, J. B. Ganey, A. Mansberger, and G. H. Yeager.

Experimental Constrictive Pericarditis—Drs. J. P. Isaacs, B. N. Carter, II, and J. A. Haller

Hearing Tests in Pre-School Age Children—Dr. John E. Bordley

Surgical Reconstruction in Cancer of the Oropharynx and Hypopharynx—Drs. M. Edgerton, and G. Ward

Observations on the Use of Propylthiouracil in the Preparation of Hyperthyroid Patients for Subtotal Thyroidectomy—Dr. Samuel P. Asper, Jr.

Studies of Hepatic Structure and Function by Fluorescence Microscopy—Dr. Allan L. Grafflin, and Miss E. H. Bagley

WEDNESDAY AFTERNOON

*Drs. Firor and Trimble presiding*

The Treatment of Fibrosarcoma—Dr. Edward S. Stafford

The Treatment of Cardiac Arrest—Dr. Jerome H. Kay

Artificial Respiration in Surgical Emergencies—Lt. James V. Maloney, Jr.

Functional Evaluation of the Status of the Pulmonary Vascular Bed—Drs. R. L. Riley, D. Carroll, and H. T. Bahnson

Studies on the Pulmonary Circulation—Dr. Henry T. Bahnson

The Present Status of Carcinoma in Situ of the Cervix—Drs. R. W. TeLinde, and G. A. Galvin

The Anatomy of Pulmonic Stenosis—Drs. T. N. P. Johns, and G. R. Williams

Measurement of Blood Flow and Vascular Volume in Limb of Man—Drs. R. Andres, H. M. Anderson, W. Stainsby, K. L. Zierler, and J. L. Lilienthal, Jr.

The Importance of Direct Surgical Attack upon Lesions of the Vertebral Bodies, Particularly in Pott's Disease—Drs. R. W. Johnson, J. W. Hillman, and W. O. Southwick

The Effect of the Virus of Eastern Equine Encephalitis on Some Normal and Malignant Cells—Drs. Frederick B. Bang, and George O. Gey

The Role of Glucuronidase in the Study of Clinical and Experimental Cancer—Dr. Andrew G. Morrow

Experimental Causation of Congenital Skeletal Defects—Dr. P. K. Duraiswami

A Hemorrhagic Disorder Occurring in Patients with Congenital Heart Disease—Drs. Robert Hartmann, and C. Lockard Conley

Present Status of Anal Ileostomy—Dr. Mark M. Ravitch

CENTENARY PROGRAM

THURSDAY, FEBRUARY 7, 1952

*Dr. Emile Holman presiding*

Welcome—President Detlev W. Bronk

Memories of Dr. Halsted—Dr. James Mitchell

Personal Recollections of Dr. Halsted—Dr. Samuel J. Crowe

My Experiences With Dr. Halsted—Dr. Willis D. Gatch

Personal Glimpses of Dr. Halsted—Dr. Arthur M. Shipley

A Pediatrician's Chance Recollections of Dr. Halsted—Dr. Edwards A. Park

Dr. Halsted as I Knew Him in the Hunterian Laboratory—Dr. F. L. Reichert

The Coming of Full-Time to the Hopkins and Halsted's Attitude Toward the Plan—Dr. Alan M. Chesney

The Story of the Hunterian Laboratory—Dr. Warfield M. Firor

Comments on Halsted's Contributions to Arterial Surgery With Particular Reference to Progressive Occlusion of the Large Arteries by Means of the Aluminum Band—Dr. Emil Goetsch

The Impact of Halsted Upon Surgical Technique of Today With Particular Reference to His Operation for Hernia—Dr. William F. Rienhoff, Jr.

Dr. Halsted as an Anesthetist Knew Him—Miss Margaret Boise

The Story of Harvey Cushing's Appendix—Dr. Samuel C. Harvey

A Follow-up on the Jackstone Calculi Removed by Dr. Halsted—Dr. R. H. Follis, Jr.

The Fruition of Halsted's Concept of Surgical Training—Dr. B. Noland Carter

Halsted's Influence Upon British Surgery—Mr. David H. Patey, London

The Story of the Discovery of Nerve Block Anesthesia—Dr. Rudolph Matas (Presented by Dr. Holman)

Presentation of Bust of Dr. Halsted—Dr. William F. Rienhoff, Jr.

Acceptance by Dr. Edwin L. Crosby

THURSDAY AFTERNOON

*Drs. Blalock and Shumacker presiding*

Presentation of Gavel to the Society of University Surgeons—Dr. Mark M. Ravitch

Acceptance by Dr. Harris B. Shumacker, Jr., President

Television Large-Screen Fluoroscopy and Radiography (Motion Picture)—Dr. Russell H. Morgan

Congenital Adrenal Hyperplasia: The Effect of Cortisone in Treatment and Its Differentiation From Virilizing Adrenocortical Tumors—Drs. L. Wilkins, and L. I. Gardner

Experimental Cardiac Defects—Drs. J. H. Kay, D. C. Sabiston, G. R. Williams, Jr., and Alfred Blalock

Physiological Considerations of Carotid Artery Ligations—Drs. A. Earl Walker, and Herbert C. Johnson

Ligation of Hepatic Artery in Treatment of Bleeding Esophageal Varices—Dr. William F. Rienhoff, Jr.

Hemipelvectomy—Dr. Mark M. Ravitch

The Endocrine Management of Disseminated Prostatic Cancer Including Bilateral Adrenalectomy and Hypophysectomy—Dr. William Wallace Scott

Surgical Treatment of Aortic Septal Defects: Experimental and Clinical Experiences—Drs. H. W. Scott, Jr., and D. C. Sabiston, Jr.

Factors Which Modify the Benefit of Mitral Valvulotomy—Drs. E. C. Andrus, and David C. Sabiston, Jr.

THURSDAY EVENING

*Halsted Centennial Dinner*

Speakers:

Dr. René Leriche, Paris
Dr. A. O. Whipple, Princeton

SOCIETY OF UNIVERSITY SURGEONS*

FRIDAY MORNING, FEBRUARY 8, 1952

*Drs. George Finney and Edward Stafford presiding*

Respiratory Acidosis: Its Relationship to Cardiac Function and Other Physiologic Mechanisms—Dr. Fletcher A. Miller (by invitation), Dr. E. B. Brown (by invitation), Dr. Joseph Buckley (by invitation), Dr. Frederick Van Bergen (by invitation), Dr. A. O. Nier (by invitation), Dr. Allan Hemingway (by invitation), and Dr. Richard L. Varco

Cardiac Catheterization Studies of Chronic Constrictive Pericarditis—Dr. J. Gordon Scannell, Dr. Gordon S. Myers (by invitation), and Dr. Allan L. Friedlich (by invitation)

The Circulatory Effects of High Spinal Anesthesia—Dr. R. B. Lynn (by invitation), Dr. S. M. Sancetta (by invitation), Dr. F. A. Simeone, and Dr. R. B. Scott (by invitation)

The Effect on the Liver of Complete Arterialization of the Blood Supply. I. Oxygen Utilization—Dr. Roy Cohn, and Dr. Chester Herrod (by invitation)

Total Replacement of the Aortic Arch by Means of Homologous Artery Grafts and Mechanical Shunts—Dr. Creighton A. Hardin (by invitation), and Dr. Paul W. Schafer

Early and Late Effects of Vagotomy on Gastric Secretions and Motility—Dr. C. Roy Rowe, Jr. (by invitation), Dr. Keith S. Grimson, Dr. Benjamin H. Flowe (by invitation), Dr. G. Keith Lyons (by invitation), and Dr. Frank H. Longino (by invitation)

The Significance of the Sympathetic Nervous System in Acute Cholecystitis—Dr. John M. Howard (by invitation), Dr. Morgan T. Milford (by invitation), and Dr. Michael E. De Bakey

Some Aspects of the Definitive Surgery of Major Vessels—Dr. Frank Gerbode, and Dr. Emile Holman

*The papers read at the meeting of the Society of University Surgeons appeared in the August, 1952, issue of SURGERY.

Thromboendarterectomy for Arteriosclerotic Thrombosis—Dr. Edwin J. Wylie

Autogenous Transplantations of Thyroid Tissue in the Treatment of Lingual Goiter—Dr. Henry Swan, Dr. Scott P. Christensen (by invitation), and Dr. Fleming I. Harper (by invitation)

FRIDAY AFTERNOON, FEBRUARY 8, 1952

*Dr. I. Ridgeway Trimble presiding*

The Retention of Various Plasma Volume Expanders Within the Circulation of Human Subjects Subjected to 1,000 c.c. Hemorrhage. A Preliminary Report—Dr. Harold G. Barker (by invitation), Dr. John D. Elder (by invitation), Dr. James M. Walker (by invitation), and Dr. Harry M. Vars (by invitation) (Introduced by Dr. I. S. Ravdin)

The Danger of Administering Parenteral Fluids by Hypodermoclysis—Dr. William E. Abbott (by invitation), Dr. Stanley Levey (by invitation), Dr. Robert C. Foreman (by invitation), Dr. Harvey Krieger (by invitation), and Dr. William D. Holden

Concepts and Methods in the Study of Total Body Composition and Its Changes Following Trauma—Dr. Francis D. Moore

The Meaning of Blood Viscosity to the Whole Blood Therapy of Burns—Dr. William C. Quinby, Jr. (Introduced by Dr. Oliver Cope)

Studies on Cortisone. II. (a) Metabolism in Cortisone Poisoning, (b) Effects of Cortisone on Tissue Culture of Fibroblasts—Dr. E. Meredith Alrich, Dr. John P. Carter (by invitation), and Dr. Edwin P. Lehman

SATURDAY MORNING, FEBRUARY 9, 1952

*Drs. W. M. Firor and Mark M. Ravitch presiding*

Extensive Small Bowel Resection in Young Dogs: Its Effect on Growth and Development—an Experimental Study—Dr. H. William Clatworthy (by invitation), Dr. Richard Saleeby (by invitation), and Dr. Charles Lovingood (by invitation) (Introduced by Dr. Robert M. Zollinger)

Congenital Malformations of the Rectum and Anus. I. Clinical Features and Surgical Management in 119 Cases—Dr. Thomas C. Moore (by invitation), and Dr. Edwin A. Lawrence

A New Concept of the Pathology of Megaloureters—Dr. Orvar Swenson

Functional Results of Small Intestinal Segments as Bladder Substitutes following Pelvic Evisceration—A Progress Report—Dr. Eugene M. Bricker

Studies on the Regeneration of Gastric Mucosa in the Dog—Dr. William P. Longmire, Jr., Dr. John M. Beal (by invitation), and Dr. Harvey N. Lippman (by invitation)

Acute Gastrointestinal Hemorrhage of Ulceration Produced by Lesions in the Hypothalamus—Dr. J. D. French, Dr. R. W. Porter (by invitation), Dr. F. K. von Amerongen (by invitation), and Dr. R. B. Raney (by invitation)

A Critical Evaluation of the Billroth I Gastric Resection—Dr. Horace G. Moore, Jr. (by invitation), and Dr. Henry N. Harkins

The Treatment of Peptic Ulcerations by Extensive Partial Gastrectomy With Gastroduodenostomy. A Preliminary Report—Dr. Frederick P. Ross

# WELCOME

DETLEV W. BRONK, PH.D.*
BALTIMORE, MD.

A CENTURY ago this year, there was born a man who benefited countless men and women of the past, the present, and the future. Although his services and influence are world wide and timeless, it is fitting that we pay him special honor here where he lived and labored longest. And so I speak to you the William Stewart Halsted.

It is right that men should pause from time to time, as we now pause, to think of those who have lived before us. For each of us there are, unknown to others, those to whom we owe our material and spiritual welfare and our very life. There are others less obscure to whom the debt of many is more obvious because of their rich endowments. Consideration of their lives, obscure or widely known, serves to remind us of the enduring values of human thought and spirit. We are thus reminded that we, too, live beyond our times, for we also shape the future.

It is not my privilege, nor am I able, to speak of how William Stewart Halsted served the Hopkins as a builder and a teacher nor how he paved the way for the surgical treatment of countless thousands. This is for others who will follow. I can do no more, and I would do no less, than simply phrase my appreciative admiration for his tireless search for truth, his deep respect for delicate living tissues, and his restless quest for understanding of disease so that disease might be cured and prevented.

In these times, when need for action seems most urgent, I would recall the wise example of one who thought that thought should precede action, lest action be misdirected.

I would pay my highest tribute to the memory of Halsted's devotion to excellence and near-perfection. This constant effort to achieve the best by always doing better is the heritage from Halsted that we of the Hopkins prize the most. It is a heritage we share with you who have unique devotion to the furtherance of human welfare—it is a heritage of example prized by all who have faith that a better future will be created by the efforts of men and women to do the best they can do.

These are times when many Halsteds are much needed, for Halsted could not be content with mediocrity, as you will hear from those who follow.

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Read at the Centenary Celebration of the birth of William Stewart Halsted, Baltimore, Md., Feb. 6-9, 1952.

*President, Johns Hopkins University.

# MEMORIES OF DR. HALSTED

JAMES F. MITCHELL, M.D., WASHINGTON, D. C.

IN REMINISCING about my nearly ten years with Dr. Halsted, I feel that it is pertinent to tell how it all happened. In April of 1893, having finished the second year in the medical school of the University of Maryland and expecting to graduate in another year, I asked Dr. Finney if he could give me some work for the summer.

He put me in the surgical dispensary and in a few days sent for me and said, "Jim, I have a job for you which may be a great thing. Dr. Halsted is away and before leaving told me that, if his operating room head nurse should leave during his absence, he wanted a man in charge. He did not say whether the man was to be a doctor, a nurse, or what; so if you want the place it is yours, and when can you start? If you make good when Dr. Halsted returns, you will be all right. If not, you can go back to the University of Maryland in the fall."

I assured him that I was ready, so that very day I started as head nurse in the operating room, where I learned to sew and to make all the dressings and supplies.

When Dr. Halsted returned in August I proceeded to try to make good. I would meet him at the front door, take his hat, coat, and gloves, follow him on his rounds, and assist him as a nurse in all his operations in the hospital and in private operations outside.

He was very active in those days, doing several operations every day. He was a chain smoker and between operations would sit at a table in his dressing room and roll his own cigarettes. While smoking one and inhaling deeply he would roll another, and this continued until the next case was ready in the operating room. He was very particular about his tobacco and cigarette papers. One day I found that he had been through my pockets as he had forgotten his tobacco. He suggested that he did not like my papers and that I would find Cosmopolite papers much better. I immediately laid in a supply and thereafter always had Bob White tobacco and Cosmopolite papers on his table—of which he was very appreciative.

I had a rough time at first. The superintendent of nurses, Miss Nutting, naturally resented my being head of the operating room and did everything to make my life miserable. Dr. Hurd also fought it.

He called Dr. Finney in one day and said he would not allow it, saying, "I know what will happen. You will all go off some day to play ball and leave this fellow in charge of the hospital."

Dr. Finney assured him that this would not be the case as they wanted Jim on the baseball team.

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Read at the Centenary Celebration of the birth of William Stewart Halsted, Baltimore, Md., Feb. 6-9, 1952.

Dr. Hurd put me on the top floor of the Administration Building with Dr. Flexner, Dr. Barker, and Dr. Mall, but would not give me any furniture for my room. I got a bed from one of the wards and some wooden boxes for the rest of the furniture. Berwick Lanier provided decorations in the shape of many-colored paper shaving balls suspended from the ceiling by means of spit balls, which greatly amused Dr. Barker. This lasted until Dr. Halsted came home when, after an inspection of the room with Dr. Mall, he immediately got for me a complete set of mahogany furniture.

He went to Wilkes-Barre that first year to do a Kraske operation on a patient of Dr. Guthrie. He took with him Dr. Finney, George Blumer, and myself. His instruments, during the operation, were kept in carbolic acid solution in glass dishes and there were large glass dishes for soaking the hands and arms in bichloride solution, so that two trunks had to be taken along for all the apparatus—much to the discomfort of the railroad baggage men who would swear at those "glass trunks." The trip was a complicated one. Dr. Halsted said he had engaged a nurse to go with us. We were to meet him at the station. Blumer and I were wondering who the nurse would be, for there was only one woman in the waiting room—an old person huddled up in the corner wrapped in a shawl.

Dr. Halsted arrived just before train time and walked over and said to the old woman, "Come along."

On the way up he said to Dr. Finney, "Finney, I am tired of these young, frivolous, talkative nurses, and I have brought a good, quiet, settled old-fashioned nurse."

Next morning we stopped somewhere on a mountainside to change trains. Dr. Halsted produced a bag with sandwiches and apparatus for making coffee and we all had breakfast, which he served. Finally we arrived in Wilkes-Barre and prepared for the operation. The old nurse immediately disappeared and we were assisted by a young 18-year-old nurse of Dr. Guthrie's—a Miss Weir.

The operation was hardly started when the old nurse dashed in and whispered something in Dr. Halsted's ear. He said very politely, "Yes." A little later when things were not going so well, she appeared again and whispered. This time he answered rather sharply, "Yes." Finally, when Dr. Halsted found he could not remove the growth and we were all tied up retracting, in she came again and whispered to him.

He said, "You shut up and get out of here," and that was the last we saw of her.

That night, coming down on the train, Dr. Halsted was sitting in front of Dr. Finney and me looking rather downcast, when he turned to Dr. Finney and said, "Finney, I am not as much stuck on that old woman as I was. She talks too much." I never heard him ask for an old-fashioned nurse again.

Dr. Bloodgood was the resident surgeon in 1893. Dr. Halsted often left him to close the wound at the end of an operation and Bloodgood would continue to operate for an hour or more, especially in breast operations.

One day that first summer, Dr. Halsted was doing an abdominal operation on a very important private patient. He left the wound to be closed by Bloodgood. In a few minutes Bloodgood was called out and turned it over to Chauncey Smith, a rather brusque individual, not too fond of work.

As soon as Bloodgood was out of the room Smith turned to me and said, "Here, you damn nurse, sew up this belly," and he also left.

I had hardly started when in walked Dr. Halsted. He seemed surprised but did not say a word. He came up to the table and watched every stitch put in.

When I had finished he said in a very quiet way, "Mr. Mitchell, do you close all the abdomens now?"

I said, "No, sir, not all of them."

He said nothing more, though I was sure I would be kicked out of the hospital immediately.

By the end of the summer of 1893 I felt that I was making good, for as soon as Dr. Halsted came in he would call for "Mr. Mitchell," as he always called me until I graduated, when it became "Mitchell" and later on, "Jim." He would not use iodoform gauze or collodion unless I had made them, and I had to hand him everything when he was making a dressing. I tried to anticipate his every wish and Dr. Henry Thomas used to say that I could tell by the wrinkles on the back of Dr. Halsted's neck just what he wanted.

In the fall there was great excitement about the opening of the medical school. I did not apply, as I expected to go back to the University of Maryland.

One day Dr. Halsted said to me, "Mr. Mitchell, I have taken the liberty of entering you in our first class and I have arranged with Dr. Mall, Dr. Howell, and Dr. Abel that you may attend classes when you wish, so you can go on with your work in the operating room."

So, for the next two years, I remained as head nurse in the operating room doing double work until I lost fifty pounds and was ordered away by Dr. Osler. I was succeeded by Dr. William Cousins of Portland as head nurse in the operating room. In the fall I came back as student intern and remained until my graduation in 1897. After a short service on the medical side I returned to the surgical staff as first assistant under Cushing. Nothing had ever been promised to me by Dr. Halsted after he entered me in the medical school until Cushing left to go abroad in 1900.

I was walking down the corridor with Dr. Halsted when he turned and said, "Mitchell, Cushing is leaving tomorrow and I have taken the liberty of naming you as his successor. What do you say?"

I said, "Dr. Halsted, what should one say when he has handed to him in the corridor what he has been working for for seven years?"

"Don't say anything. Just get to work," was his reply.

Dr. Finney had much the same experience. After his talk with Dr. Halsted the day the hospital opened he never talked with him again about his position, although he advanced steadily from Dispensary Surgeon to succeed Dr. Halsted as Professor of Surgery.

In those early days, as always, Dr. Halsted was exacting in observance of strict operating room technique. Cleansing of the hands was a strenuous ritual. First, the hands and arms were scrubbed for ten minutes with a stiff brush and green soap, then they were soaked in a saturated solution of potassium permanganate, then in a saturated solution of oxalic acid, and, finally, five minutes in 1:1,000 solution of bichloride of mercury. Neither the operator nor assistants wore caps, masks, nor gowns. The assistant who handed instruments wore gloves, for after the instruments had been boiled they were placed in 1:20 carbolic acid and distributed in dishes of 1:40 carbolic acid, but no other gloves were used. The operating room was very plain. It had a wooden floor and the operating table was a wooden trough on which was a heavy oak board. The board was carried to the ward and the patient strapped on it and brought to the operating room. There were no electric lights and no elevators in the hospital and getting this board up and down from Ward E on the third floor was a real job.

Dr. Halsted was quick to see any break in technique and to correct the offender, never harshly but always impressively. In the careful and gentle handling of tissues it was the same. His reproofs were convincing, sometimes sarcastic, but never ugly. One day, after he had finished operating, George Blumer and I asked him if we might get off for the afternoon to go to a football game.

He said, "Why certainly, never let surgery interfere with football."

Blumer and I went back to work.

Another time I was handing sponges with one hand and retracting with the other, not being able to see the retractor, when Dr. Halsted said in the gentlest way, "Mr. Mitchell, I saw an operation once when an assistant was holding a retractor on the celiac axis. He tried to chin himself on the retractor. The axis broke and the patient bled to death."

I said, "Yes, sir, I will be more gentle." In the nearly ten years that I worked with Dr. Halsted I cannot recall that he ever spoke sharply to me or reprimanded me harshly.

In his dress he was immaculate and most particular. His shirts, fifty at a time, were sent to England to be laundered. He nearly always wore a high silk hat.

When I showed him the picture of The Fates—himself, Dr. Osler, and Dr. Kelly—taken in 1894, he looked at it for some time, seemingly quite annoyed, and then remarked, "Mercy! my hat never looked like that."

I explained that the picture was cut out of a large group photograph I had taken, which accounted for the hat looking rough. He seemed quite relieved.

In 1905, he came to Washington to be godfather for my oldest boy. The christening was to be in the afternoon.

He arrived in the morning, saying, "Jim, I did not know what color gloves I was expected to wear, so I have brought four pairs of different colors."

We all admired the spirit of comradeship that existed among our professors, whom we considered old men although they were all in their forties. Dr. Osler was the particular tease. During operations three Arnold sterilizers were on a table beside the nurse—one containing sponges, another towels, and the third dressings.

Dr. Osler would come in and ask, "Halsted, what are you doing?"

He would put his cane or umbrella in one sterilizer, his gloves in another, and his hat in the third, and then walk around to get a good view of the operation.

Dr. Halsted would say, "Osler, will you never grow up?" and that was all.

When Dr. Halsted was first using silver foil as a dressing he was taking a visiting English surgeon through the wards, demonstrating the beautiful healing under silver foil. In one ward there was a row of hernia cases. The hernia dressing consisted of a layer of silver foil and paper over the incision with a subcutaneous continuous silver wire suture twisted over a gauze pad. Over this was a large gauze dressing covered with a plaster of Paris cast reinforced with thin wooden splints from the armpits to the knees. This was not touched for twenty-one days. Then the cast was removed, the wire suture cut at one end and withdrawn, and the paper lifted up showing a well-healed incision. Dr. Halsted had showed several of these to the astonished and admiring Englishman. They came to a patient whose chart showed an absolutely normal temperature from the day of operation. When the suture was withdrawn the wound fell apart and several ounces of pus flowed out—the result of a broken-down blood clot. I wondered what two gentlemen would do under such conditions. Neither said a word, but passed on to the next patient.

I went with Dr. Halsted one day to see Dr. Kelly do an operation he had devised for the cure of inguinal hernia. This consisted of implanting a marble in the inguinal canal to hold back the sac. Dr. Halsted intently watched the operation without saying a word.

Then when Dr. Kelly asked him what he thought of it, he said, "Just one question, Kelly, does the marble have to be green or would a pink one do just as well?"

The working of Dr. Halsted's mind was to me always wonderful—he was always looking ahead. He noticed that dentists found that gold as a filling in teeth lasted better than anything else and wondered if gold had some bactericidal property. So he started Dr. Meade Bolton to work in the laboratory. Bolton found that if a piece of sterilized gold was dropped on a plate culture, organisms did not grow around the gold, so Dr. Halsted started using gold foil as a dressing for fresh incisions. This was expensive, so other pure metals were tried by Bolton with similar findings. Dr. Halsted shifted to copper and then to silver. This was the origin of his silver-foil dressing.

I was assisting him one day, trying to put a shot in the common bile duct of a dog to simulate gallstones. His broad thumb obscured the opening in the duct and the shot was lost.

After many unsuccessful attempts, he asked, "Can you think of any way to get this shot in the duct?" I suggested that if it was stuck on the end of a long hat pin it might work.

"Who ever heard of sticking a hat pin into a shot?" said Halsted. However, it did work, to his great delight.

In writing the protocol he described the pin and shot procedure and went on to write, "Why would it not be a good thing to have made little hammers with long handles to facilitate the placing of sutures and removal of the hammer before tying the sutures in the common duct, or a collapsible rubber bag used in the same way for intestinal suture?" These were both made and used for a time.

He was a prolific letter writer but never dated his letters except by the day of the week. He would write a letter always in longhand, often with a quill pen, and send it over to the hospital by special messenger at night, usually marked "urgent" when he was certain to be at the hospital next morning. Usually, the most important part was in the postscript, sometimes two or three postscripts, for example, "Please have Mr. So-and-so for operation tomorrow. P.S. Please send me by messenger a large package of silver foil. I am sending it to Von Bergmann. He is greatly interested and wishes to try it."

There had never been a secretary-stenographer assigned to the surgical department until I became resident. With a salary for the first time in my life, forty-one dollars and sixty-six cents a month, I felt very rich and engaged at my own expense Miss Reich, an excellent stenographer and trained her so as to be ready for Dr. Halsted on his return. He was delighted with her and immediately engaged her as his private secretary with a contract for a year. In a few weeks she came to me and asked if I could get her another place as Dr. Halsted did not need her any longer. I turned her over to Dr. Hurd with whom she worked for many years. I asked Dr. Halsted what the trouble was.

He said, "Mitchell, she would use perfume and I told her I objected to it, that it was distasteful to me, and what did she do but change the brand; so I gave her a year's salary and let her go."

He then went into a long dissertation on the expense of keeping clean—soap, towels, etc.

Bloodgood once ordered me to make a complete blood count: leukocytes, red cells, and hemoglobin, every half hour on a patient of his. I protested that it was impossible for one man to do this and clean his instruments between counts. Dr. Halsted, hearing the argument, asked Bloodgood if he had ever made a blood count.

When he acknowledged that he had not, Dr. Halsted said, "Neither have I."

His look and silence were often worse than a reprimand. He conceived an operation for cerebrospinal meningitis which was to fit into the skull a watertight cannula, then do a spinal puncture and irrigate through with an

antiseptic solution. It got noised about and there was a great crowd assembled in the amphitheater. He had placed the cannula in the skull and the patient was turned on his side.

Dr. Halsted whispered to me, "Mitchell, I have never done a spinal puncture. Will you do it?"

With the brass of ignorant youth I took the needle and with luck introduced it at the first attempt.

When the spinal fluid ran out he said, "That looked very easy. How many have you done?"

When I answered, "This is my first, sir," I shall never forget the deep sigh and the look he gave me as much as to say "you nervy little fool."

I think his shyness had a great deal to do with it. He never liked a crowd. When the American Surgical Association met in Baltimore in the nineties he was to demonstrate his breast operation.

When the operation was about to begin he said, "You operate, and I will stand by."

With his assistants, even the youngest, he was always courteous, never familiar, and the same with his students.

He occasionally told me some interesting experience he had had, for instance, the description of a dinner with Von Bergmann: "There were eight or ten different wines. Ordinarily I am not much of a drinker, but that night I just didn't care and tried all of them. Before the dinner was over Mikulicz on one side of me and Schede on the other both had their arms around my neck begging me to come and live in Germany."

He was very partial to anything German so that, in 1896, he appointed as resident surgeon, over the heads of everyone, one of Von Bergmann's assistants, whom he had never seen—a Dr. Werkmeister, an enormous man, his face well scarred from student duels. Dr. Halsted went away and, as usual, left Dr. Finney to break him in. Dr. Finney did not have the same confidence that Dr. Halsted had in him and cleaned up to watch him do his first operation—a strangulated inguinal hernia. After much rough surgery he eventually cut into the intestine and holding up a finger said, "Ah! feces," which got him oriented.

Dr. Finney pushed him aside and finished the operation. That was Dr. Werkmeister's last operation. He did not stay long, but before leaving challenged little Cousins, the head nurse, to a duel, which Cousins politely declined.

Another thrilling experience Dr. Halsted described to me. He was driving alone in an open buggy with one horse in the mountains of North Carolina. They came to a stream which ordinarily was easily forded but that day was a raging torrent and the horse and buggy were swept downstream.

"I was afraid the little horse would be drowned and I was very fond of her; so I jumped out and swam alongside of her and steered her into shore."

He was a great believer in heredity and breeding. When our class first came under his observation he asked me to point out Tom Brown.

"He comes of good stock and I want to know him."

This request was repeated many times and in the fall of 1896, as we were entering our last year, he asked me if I would not like to come on the surgical staff as all positions were being held open for our graduates. I reminded him I had been on the staff for some time.

Then he said, "Do you think Tom Brown would like to come on?" He was greatly surprised when I told him that Tom had also been on the surgical staff for some months.

I never felt that he could pick men until they had shown their ability. Hugh Young came to the Hopkins in 1896 for a postgraduate course. He did good original work that first year on the bacteriology of cystitis and in 1897 asked Dr. Halsted if he would give him a position on the surgical staff. He was flatly but politely refused and Young went back to work. A year later he again applied, with the same result. When Dr. Halsted went away in the summer Dr. Finney appointed Young and when Dr. Halsted returned he found him installed as a member of his staff, and he always felt that he had discovered Young. It was the same with Follis who, when he graduated, was not given any hospital position. When I became resident I telegraphed him in San Francisco to come on and I would give him work. Dr. Hurd would not give him a room and for a time, as my first assistant, he slept on a window seat in my room. Dr. Halsted became very fond of Follis who followed me as resident and eventually did the first operation for Dr. Halsted's gall bladder trouble. Almost the same thing happened with Watts. Staff appointments were often made without Dr. Halsted's dictation, especially during his absence in summer, but once the incumbents had proved their worth everything was done by him to encourage and help them. I know of only one instance—that of Sowers—in which he promised anything in advance. Once an assistant showed great interest in any subject he was given free rein, as one year he turned over to me all operations for tubercular glands of the neck of which, in the early days, there were great numbers.

He was very dependent upon his assistants and no matter how many were around the table he would find something for each one to do. He abhorred suggestions but welcomed anticipation of his wants, and his resident was always expected to have ready answers to any questions he might ask. Cushing and I, one opposite him and the other beside him at the table, used to take great delight in carrying him through a long breast operation without letting him put on a clamp or tie a vessel or ask for anything, and on these days his keen appreciation was apparent.

His knowledge of anatomy was perfect and his dissections beautiful. He was not a rapid doctor, but rather slow because of his extreme care in hemostasis and gentle handling of tissues.

Only twice did I ever see him give up in an operation. He was operating upon a very important private patient for glands of the neck. While working low down there was sudden profuse arterial bleeding. I put my finger on the offending vessel.

"What was that?" asked Dr. Halsted.

I said, "I think it is the vertebral and I can put a clamp on it."

"Don't you dare, I will resect the clavicle and tie the subclavian."

When he had taken out the inner half of the clavicle and put a ligature around the subclavian but had not tied it, he said, "Do you really think you can put on a clamp? All right, Follis, you help him."

He backed away from the table and when the vessel was ligated he said, "Now you finish the operation," and taking off his gloves left the room, and never saw the patient again.

One day in 1899, three surgeons, Dr. Mallock of Toronto, a former assistant of Lord Lister, with Dr. Farrar Cobb and William Conant of Boston, arrived and said they would like to see Dr. Halsted operate next morning, so I called him on the telephone. He asked what there was in the ward. I told him there was a very large thyroid.

"That's fine. I'll do it under local anesthesia."

Dr. Halsted arrived and asked the questions I had expected: "How many thyroid operations have we had since the hospital opened? Has Cushing done any? Have you ever done one?"

As a matter of fact, there had been only seven in those first ten years, of which Cushing had done one and Dr. Halsted six.

He then proceeded to give a most interesting talk and concluded with the remark, "This is the largest thyroid I have ever seen."

The anesthetic was Schleich's solution of cocaine and morphine. Dr. Halsted started the operation but, always fearful of cocaine, did not use enough of the solution and in a few minutes the wound was bristling with clamps and the patient struggling.

Dr. Halsted looked up at me and with a deep sigh said, "Mitchell, I have an awful headache."

I had the nurse get him a cup of coffee. He went in the next room to drink it. We got the clamps off and the patient quieted down and he returned, changed his gloves, and started in again. In a few minutes there was the same thing and the muscles not yet divided.

He said, "My headache is worse. I am going out and rest a while."

This was repeated again and as he left the third time he said, "Now this time you go ahead with the operation. Good-bye."

Bloodgood and Cushing were both away and my first assistant, Staige Davis, had just graduated. We finished the operation and I explained to the visitors that Dr. Halsted was subject to these headaches.

The visitors stayed on for dinner and that evening while we were at the table a messenger arrived with a package and a letter from Dr. Halsted.

"Dear Mitchell. I telephoned and found that you finished the operation and that your patient was all right. Some day you will know what it means to have an assistant in whom you have confidence. I hope you will enjoy this bottle of old Madeira."

A few days later in a letter giving directions about a patient there was a postscript as usual: "I was very sorry to leave you so short handed the other day, but my head ached so severely that I became sick at my stomach and was afraid to remain."

I heard him allude to cocaine only twice. Once we were going through the wards and came to a man who was very restless.

He asked what was the matter with him and when I told him I had operated that morning under cocaine for a very large hernia he said, "Give him morphia. If you knew how terrible the suffering is with that restlessness after cocaine, you would not stint his morphia."

The second allusion was in 1904 when, at the opening of his new operating room, he operated with what Cushing called his all-star team of assistants. He asked each of us to talk for a few minutes. In my talk on local anesthesia I alluded to some of his papers written in 1884-1885 which I had unearthed in New York.

He said, "Where did you get that awful stuff? I was not in very good shape then."

I feel that the ten years I was associated with Dr. Halsted as nurse, student, intern, assistant, and resident surgeon were the happiest of my life and I think of him always with extreme admiration and deepest affection—a cultivated and refined gentleman and a great surgeon.

# PERSONAL RECOLLECTIONS OF DR. HALSTED

SAMUEL J. CROWE, M.D., BALTIMORE, MD.

IT WAS by the purest chance that I came to know Dr. Halsted the man, and not merely the surgeon. To make this clear I will have to retell the story of the sick horse.

At the end of my first year in the Johns Hopkins Medical School, June, 1905, I asked two friends to join me on a camping trip in the North Carolina mountains. We hired a wagon and two Percheron horses, and for several weeks drove aimlessly through the mountains with no maps or guides of any kind. At night we slept in the wagon and tied the horses to trees. This was an unusually cold and rainy summer and one morning when I went to feed the horses I found one of them standing with front legs far apart, head hanging, and so stiff that it was difficult to get him to move. Here I was, a relatively penniless medical student, deep in the mountains far from any railroad or town, with a hired horse that looked as if he would never walk again. Without waiting for breakfast, a shave, or a wash I mounted the well horse and, moving at a snail's pace, led the other down the road in search of a barn and some advice about what to do for a sick horse. Finally, I came to a side road and saw a log cabin in the distance. An old mountaineer was sitting on the porch. He agreed with me that this horse should be under cover and should be seen by a doctor, but he had no other suggestions. It was quite evident that his stable was far too small to house such a huge animal. I was becoming more worried every minute. To my intense irritation the man seemed far more interested in learning where I came from, where I was going, and why I was driving through the mountains than he was in helping me with the horse. It finally dawned on me that he suspected I was a revenue agent hunting for illicit stills. I answered his questions as patiently as I could and finally told him I was a medical student at the Johns Hopkins in Baltimore and was here on a vacation. Immediately he became more friendly and informed me that Dr. Halsted lived about two miles up the road, that he also came from Baltimore, and that his barn was the only one in these mountains large enough for a horse of this size.

Leaving the sick animal standing in the road I soon arrived at Dr. Halsted's estate, High Hampton, which consisted of 3,000 acres of woodland, two small houses, a beautifully kept lawn, row upon row of the most gorgeous dahlias, and a large barn. It was the barn that interested me most. I subsequently learned from Dr. Halsted that he had been collecting rare and beautiful varieties of dahlias for many years. This garden was his chief delight. My knock on the door of the larger of the two houses was answered by a maid in a dainty white uniform and cap. She said Dr. Halsted was out walking on the place. I sat on the porch feeling very uncomfortable because of my dirty clothes and dishevelled appearance. This feeling was accentuated when Dr. Halsted appeared

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about an hour later in spotless white flannels. I told him who I was and why I had called on him. Instead of sending his stableman with me, he immediately had his buggy brought around and insisted that we drive down and look at the sick horse. That impressed me as being exceedingly kind and polite to a first-year medical student he had never seen before, but what impressed me most was the care and patience with which Dr. Halsted examined the horse after we got there. Disregarding the muddy road he got out of the buggy and for fully thirty minutes palpated every joint and muscle, and observed very minutely the horse's gait as I walked him up and down the road. All this time the mountaineer and his family leaned on the fence and looked. They were not surprised at what they saw. For years the natives had been accustomed to call on Dr. or Mrs. Halsted when their cow, their horse, or any member of their family was sick. The Halsteds never refused, because doctors were few and far between in those mountains forty-seven years ago. Dr. Halsted had always been a student of anatomy, and the number of sick and injured animals he was asked to see during his vacation period in the mountains stimulated in him an interest in veterinary medicine. He had acquired textbooks and the necessary instruments, and each year had become more and more interested in the treatment of sick and injured horses and dogs. Mrs. Halsted, who had spent most of her life on a plantation and was accustomed to seeing animals operated on and treated by the crude methods of the natives, was greatly amused at some of the Professor's exploits in the field of veterinary medicine. At any rate Dr. Halsted diagnosed my horse's disability as muscular rheumatism. It is true he advocated bandaging the horse from head to foot with oiled silk, which of course was not available, so he had me go to the country store and buy oilcloth such as is used on kitchen tables. After a week in a box stall, rubbed daily with liniment and bandaged with strips of oilcloth, the horse recovered and I went on my way.

That week at High Hampton initiated a change in my attitude toward medicine. Every morning Dr. Halsted examined the horse and then, sometimes accompanied by Mrs. Halsted, we would walk through the dahlia garden inspecting the flowers for insects or sit in chairs on the lawn and talk. Looking back on those days I realize what this chance meeting with Dr. Halsted has meant to me. He pictured medicine as a living, growing, constantly changing and most fascinating study. To me, this was a new concept and when I returned to the medical school a few weeks later, it was with enthusiasm and joy.

Dr. Halsted was a most gracious and charming host, always full of conversation, sparkling with wit, and interested in a thousand things. Others have pictured him as shy and aloof. This is not the Dr. Halsted I knew. He and Mrs. Halsted when at High Hampton delighted in driving along the mountain roads with Nip and Tuck, the older dachshunds, in the buggy with them, while Otto, Mädchen, and the setters ran along beside them. The dachshund was Dr. Halsted's favorite dog.

In a letter dated Jan. 3, 1903, to the young son of one of the University professors he said: "You must come and see our new dachshund; she is a great beauty, a brunette with a lovely complexion and all the graces of her sex."

When on these drives they met any of the mountain people, Dr. Halsted usually stopped and talked, often I am afraid in language quite beyond their comprehension. He was extremely popular, however, having treated so many of them for minor injuries and sending the more seriously ill to the Johns Hopkins Hospital at his own expense. The whole countryside rang with praise at the miraculous thing he did for one patient. This man was working with a band saw and had several of his fingers cut off. The bleeding was controlled with a handkerchief tied around his arm and tightened with a stick. The severed fingers were wrapped in a piece of newspaper and the man was brought to Dr. Halsted with the request that he sew them on. This he did, and the hand healed without infection or postoperative edema. This was many years prior to his experiments showing that wound infection, and not solely blocking of lymphatics and veins, was the cause of swelling of the arm following radical operation for carcinoma of the breast.

During the next seventeen years it was my privilege to see much of Dr. and Mrs. Halsted in their house at 1201 Eutaw Place. There I learned the story of how rubber gloves came to be used in the operating room.

Mrs. Halsted was Miss Caroline Hampton, the niece of Wade Hampton of South Carolina, who, before the Civil War, was the largest cotton-grower in America. During the war he was elevated to the rank of Lieutenant General and was on General Lee's Staff. Her father, also an officer, was killed in the battle of Brandy Station in Virginia. After the war the Hamptons were desperately poor and Miss Caroline determined to become a nurse. She graduated at the New York Hospital in 1889 and was appointed head nurse in the surgical division of the Johns Hopkins Hospital when it was formally opened in May of that same year. In those days and for approximately twenty-five years thereafter, it was the custom on the surgical service to scrub the hands and arms vigorously with soap and water and then soak them for ten minutes in a long, narrow basin filled with a 1:1,000 mercuric chloride solution. This technique produced such a marked dermatitis on Miss Hampton's arms and hands that Dr. Halsted asked the Goodyear Rubber Company to make, as an experiment, two pairs of thin rubber gloves with gauntlets. These gloves proved so satisfactory that additional gloves were ordered. Within a few months not only the nurse, but also the assistant who threaded the needles and passed the instruments began to wear gloves. For several years after this the operator and first assistant wore gloves only for operations on joints or the central nervous system. It was realized that gloves improved the aseptic technique, but for a long time it was thought that they impaired the sensitivity of touch. I have seen Dr. Halsted and senior members of the staff remove their gloves to palpate a nodule in the breast or to feel for stones in the bile passages. The younger members of the staff, however, grew up with gloves and when they became residents they rarely, if ever, removed a glove to improve the sense of touch.

Miss Hampton was head nurse in the operating room for only one year. She and Dr. Halsted were married in June, 1890, and went to the Hampton family hunting lodge in the North Carolina mountains for their wedding trip. This is the property that Dr. Halsted later bought and called High Hampton.

Both Dr. and Mrs. Halsted had a keen sense of humor, and one day over a cup of tea they told this amusing incident of their wedding trip. Dr. Halsted had been born and bred in New York City and occasionally had ridden a horse in Central Park, but Mrs. Halsted was an expert and enthusiastic horsewoman.

She was anxious to show him the place, and a day or so after their arrival at the hunting lodge the two of them were riding along a trail in the woods when Mrs. Halsted suddenly pulled up her horse and pointing with her riding crop exclaimed in the greatest delight, "William, there is a rattlesnake, get down and kill it."

Looking at me with a twinkle in his eye Dr. Halsted said, "There I was alone in the mountains with this comparatively strange woman, and she wanted me to get off my horse and kill a rattlesnake. She was terribly disgusted when I refused."

They were a devoted pair, although entirely different. His chief interests were surgery, research, and his medical books. He was not interested in poetry or general literature. His recreations in Baltimore were the collection of antique and beautiful furniture, rugs, china, and silver, which filled his house on Eutaw Place and at High Hampton. He liked the theater, but cared little for art or music. When in North Carolina his chief interests were dahlias, the study of comparative anatomy and diseases of animals, and astronomy. He had a beautiful reflecting telescope and showed me many charts he had made of the moon, the planets and their satellites, and some of the fixed stars. Mrs. Halsted's chief interests in life were to make everything comfortable and pleasant for Dr. Halsted, and to interfere as little as possible with his work. Their Baltimore home was a large, three-story brick house. Mrs. Halsted's quarters and the guest rooms were on the third floor. He and his books occupied the second floor. The house was largely heated by open wood fires. Dr. Halsted liked only hickory and white oak. He also was very fond of fresh guinea eggs for breakfast, said they had a gamey taste. On many occasions Mrs. Crowe and I have driven Mrs. Halsted far into the country around Baltimore searching for just the right kind of firewood and the right kind of eggs for the Professor. Mrs. Halsted always went to High Hampton several weeks before the date of Dr. Halsted's arrival in order to supervise the planting of the dahlias and the vegetable garden, and to get the lawn in perfect condition. She was a very practical and efficient woman. In a letter written in 1914 she mentions that she had sold a calf to one of the mountain women for a stove, four pairs of knitted wool socks, a dollar and ten cents, and a half dozen eggs. The socks were sent to the soldiers.

Dr. Halsted's study was on the second floor adjoining his bedroom. A wood fire always burned in this room, which was filled with the books and journals he was using at the time. His library was on the first floor, but was used only to house the books. He rarely went out, but often worked far into the night on a paper he was writing or in preparation for a clinic or ward rounds the following day. He consulted all the literature, both ancient and modern, that bore on the condition to be brought before the class. In thus preparing for a clinic he would sometimes become so engrossed in making innumerable notes about new ideas for diagnosis, treatment, or some research project that he lost all count of

time. The following day he would be so fatigued that he would call his resident at the hospital and ask him to take over the class and the operations scheduled for that day. When Dr. Halsted did give a clinic, many of the questions he asked the students were far over their heads, and often left the house officers without an answer. This method of teaching aroused many of us to think and to search more deeply for the answers than we otherwise might have done.

Few of us realized at the time how much we gained from association with this man. He was tireless in tracking down every bit of information that would help to solve a puzzling problem. Also he took every opportunity to insist to students and house officers that the work of others always should be recognized and given due credit. Never would he publish a clinical report or experimental study until he was sure that the fundamental facts were correct. He often said that all men are fallible in their interpretation of the facts they present, but the facts themselves must be correct. That was his creed.

1801 EUTAW PLACE

Dear Mrs. Rowland:

I have only this moment
heard of your appalling sorrow
If there is anything in the world
that I can do please please
tell me. Every moment of my
time is at your disposal.
With deepest sympathy and
affection I am
Faithfully yours
Tuesday W. S. Halsted

His depth of feeling, loyalty and devotion to his friends is shown in a letter to Mrs. Henry A. Rowland, the widow of the distinguished Professor of Physics at the Johns Hopkins University.

# MY EXPERIENCES WITH DR. HALSTED

WILLIS D. GATCH, M.D., INDIANAPOLIS, IND.

ONE morning at ward rounds, when I was a senior medical student, I met Dr. Halsted for the first time. I then presented the case of a Negro who had an aneurysm of the celiac axis. Dr. Halsted operated twice for this. The first time he constricted the aorta just above the diaphragm with an aluminum band; the second time he constricted it with a band placed just below the renal arteries. I saw him daily during the postoperative course of this patient. I had intended to take an internship in medicine, but at his invitation I took one in surgery. For the next four years I assisted him for about three months every winter in his experimental work.

Dr. Halsted was then 55 years old and at the top of his professional career. I have always fancied that his physical appearance was an accurate reflection of his character. It was singular but distinguished. He had an athletic build, and was of medium height. His complexion was ruddy. His head was finely shaped. His hair was scanty. He wore a mustache and a goatee, which were already gray. He had powerful hands with short thumbs. He dressed extremely well; wore gloves and carried a cane. He was so shortsighted that he could not easily recognize people when he passed them. He was uneasy in a crowd and did not use first names. Dr. Finney, Dr. Young, Dr. Bloodgood, Dr. Baer and Dr. Cushing frequently had lunch with the surgical residents and interns; Dr. Halsted, rarely. He hated familiarity and feared its consequences. I never heard him say anything but good of his colleagues, though I noticed that his relationships with none of them seemed to be intimate—Welch, Mall, and MacCallum excepted. He always thought before he spoke. Conversation with him was difficult and even dangerous. He had a Boswellian trick of feigning a great interest in the subject under discussion along with the most pathetic ignorance of it. With this he quickly pumped his victim dry. I saw him use it many times on visitors in the laboratory and I am sure that none of them—not even MacCullum—detected it. I think his habit of managing a conversation in this way together with his whimsical sense of humor gave him the reputation of being as incompetent as a child in the ordinary affairs of life. MacCullum had this opinion and mentions in this connection that Dr. Halsted sent his laundry to London, not knowing that he could have it done in Baltimore. Maybe the Professor spoofed someone by pretending not to know that clothes could be washed in Baltimore. Members of the resident staff in surgery frequently discussed his characteristics. The one of these about which they all agreed was that he could not easily be fooled about anything.

Read at the Centenary Celebration of the birth of William Stewart Halsted, Baltimore, Md., Feb. 6-9, 1952.

Dr. Halsted was very mindful of the exact meaning of words and took great pains with his writing. He rewrote his papers many times and they are models of clear exposition. He never rushed into print and did not force his residents to grind out papers. One spring he invited me to be his guest at a meeting of the American Surgical Association. He had worked for weeks on a paper which he intended to read at this meeting. To my astonishment he did not read it but talked on a totally different subject. He must have had last-minute doubts on the truth of its contents. He was very cautious about making positive statements. Once after he had read a paper I was preparing, he advised me to learn the value of the word "perhaps."

"If you put 'perhaps' before a statement," he said, "and the statement turns out to be true, you will get credit for making it; if it turns out to be false you will not be blamed."

Early in January every year he would call me to his office to inform me that he was ready to start his experimental work in the Hunterian laboratory. I would then hire a Negro boy to procure dogs and instruct him on how to etherize them. I would collect the necessary instruments and dressings and have them sterilized in the operating room. Dr. Halsted kept his own notes on the experiments. Though he was the leading experimental surgeon in the United States, this was all the setup he used. He would have been unable to work with the elaborate organization of the present-day experimental surgeon. While I assisted him he worked on thoracic surgery, vascular surgery, and the technique of aseptic anastomosis of the intestine. He used intratracheal insufflation successfully on dogs but he never published anything about it. He almost invented the simple devices for controlling respiration which make possible the chest surgery of today. When he applied an aluminum band to the thoracic aorta, he used a positive pressure helmet to inflate the collapsed lung. He thought that up to that time no intrathoracic operation on the human being had been done with any kind of device for control of the respiration which could not have been done equally well without it.

He applied aluminum bands to large arteries of dogs with fairly successful results. He found, however, that the clinical use of these bands was dangerous. He cracked a sclerotic abdominal aorta with one of them and was hardly able to get the patient off the table alive. In this crisis his surgical calm was undisturbed.

"Act like surgeons," he said to his trembling assistants.

He performed successfully sutures of blood vessels, but while I was with him he never had the opportunity to suture a human blood vessel. He thought he had conquered infection in all operations except those on the intestine and his failure to overcome it in these troubled him. For years we worked on this problem. Dr. Harry Kerr had just published an account of his ingenious "basting stitch" technique for aseptic suture of the bowel. Dr. Halsted did not regard this as aseptic. I suggested the bulkhead technique to him and he worked to perfect this almost to the time of his death. He would have thought that sterilization of the bowel with drugs took an unsportsman-like advantage of the bacteria.

Dr. Halsted had the reputation of being a very slow operator, though by present standards he would be regarded as a very fast one. He always knew where he was and what he needed to do and kept on doing it. It requires time to accomplish surgical workmanship as excellent as his. Surgery, in his opinion, was no calling for weaklings. He rebuked his assistants with dreadful sarcasm without raising his voice. He did not enjoy himself at the operating table. The performance of routine operations vexed him.

"Surgery," he said, "would be delightful if you did not have to operate."

His conduct in the laboratory was in total contrast with that of the operating room. Here he obviously enjoyed himself and was at ease. He was always ready to consider suggestions and was always ready to change his opinion when the facts demanded it. He was interested in establishing the principles of surgery and in developing the technique of various operations. Once he had accomplished these purposes, he was entirely willing to let others make clinical application of his work.

I have been often asked whether I think Dr. Halsted conquered his cocaine habit. I never saw him do anything that would make me think otherwise. I knew nothing of this habit until I read MacCallum's *Life,* nor had anyone I knew while at Hopkins ever heard of it.

I close with a paradoxical conclusion. Dr. Halsted had the devotion of a long line of assistants because of his austerity and not in spite of it.

# PERSONAL GLIMPSES OF DR. HALSTED

ARTHUR M. SHIPLEY, M.D., BALTIMORE, MD.

MUCH of the influence of Dr. Halsted's teaching of surgery reached me indirectly. I entered the University Hospital at the end of my junior year in 1901 as a clinical clerk. I was student assistant on all of Dr. Tiffany's operations, and on most of Dr. Martin's. In a short time I knew that Dr. Martin was at this time the best surgeon in the hospital. He often spoke of Dr. Halsted's method of operation. The small straight hemostats were called Halsteds. The three operations in which he followed Dr. Halsted's technique were for inguinal hernia, radical mastectomy for cancer, and subtotal thyroidectomy.

Dr. Martin was cool and did not hurry. He clamped bleeding points without strangulating adjacent tissues; he was very careful not to traumatize healthy tissues. He used few retractors and very little packing. The preoperative preparation of instruments, gauze, and material for draping the wound was good. In those days we scrubbed our hands 15 minutes (by the clock), immersed our hands and forearms in a strong solution of permanganate of potassium, and dissolved this purple substance with oxalic acid. We soaked our hands and forearms in a solution of bichloride of mercury and then put on boiled, seamed gauntlet gloves wet. We wore no masks, but wore sterile gowns with caps. There was little talking. Dr. Martin was skillful in seeing that the position of the patient on the table would give him the best possible exposure, and that the position of the table in relation to the light would permit seeing into the wound. He maintained a rigid discipline in the operating room.

There was no other operator in his class in the University Hospital, and he freely gave credit to Dr. Halsted. He had spent most of his teaching time in regional anatomy and operative surgery. I was assigned to him for three years as clinical clerk, intern and assistant resident. In 1904, I became resident surgeon and remained in this position four years.

After the First of October, 1904, I telephoned Dr. Halsted's secretary at the Johns Hopkins Hospital, identified myself and asked for a short interview with Dr. Halsted at any time that suited him. After a short interval, I was called at the University Hospital and asked to be present at a definite hour the next day. I was on time and Dr. Halsted saw me almost at once. I told him briefly that I was resident surgeon at the University Hospital, and then requested permission to attend his clinics when I could, that I could see better from the benches, that I would be in street clothes and did not wish to enter his operating room. I explained I was not applying for any graduate course of any kind. He at once gave his consent and was altogether very courteous and natural in his manner. The interview occupied five or six minutes. I do not know to this day how Dr. Martin learned to follow so many of the principles and details of Dr. Halsted's way of operating unless he learned, as I did later, by watching him operate.

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Read at the Centenary Celebration of the birth of William Stewart Halsted, Baltimore, Md., Feb. 6-9, 1952.

During my four years as resident, I sat with the students in either Dr. Halsted's or Dr. Osler's clinic when I could get away from the University, and took Dr. Bloodgood's course in surgical pathology. It was very surprising to me how closely Dr. Martin followed the details of Dr. Halsted's operative technique.

In 1910, the care of patients, the buildings, and equipment at Bay View were investigated by a committee appointed by the Supervisors of City Charities. I served on this committee and was surprised and shocked by what I saw. The committee report led to the building of a new hospital, and on Jan. 1, 1911, a new staff took over the medical care of Bay View patients. Dr. Boggs was Medical Chief and I was Surgical Chief. I remained in that position about twenty-five years.

This led to my second short interview with Dr. Halsted. He asked me if I were willing to undertake the teaching of a course in Clinical Surgery limited to twenty-five Hopkins juniors on a volunteer elective basis once a week for one trimester. I agreed, and he said it would be a pity if so much clinical material were not used in teaching. This course I carried on yearly for about twenty-five years.

During this period on one occasion I happened to have seven interesting hip joints with good roentgenograms and clear pathologic pictures, all different, which had been shown to the Hopkins juniors.

I received a telephone call from Dr. Halsted saying he had heard of this group on hip joint disease, and would I give his clinic the following week to the senior class at Hopkins. This set me back a bit, but I managed to say that I would, and would do my best. The Hopkins students did not worry me, but I had heard much discussion about the disturbing effect of Dr. Halsted on men teaching in his presence. At any rate, I memorized the histories of these seven patients and had a different intern or assistant resident bring in each patient. I gave each man two minutes to get the next patient in the amphitheater, to put the roentgenogram in the view box, and to state to the class what we thought was the diagnosis. I narrowly escaped a complete defeat in this clinic, and to this day I don't know why.

At the beginning of the clinic, Dr. Halsted introduced me to the class briefly but with courtesy and consideration. He then did something modestly and simply but which very nearly ruined me during the hour-long clinic. He walked across the amphitheater and sat at the end of the first row on my left. The only thing that saved me was that the blackboard and view box were on the right.

I am sure that he was paying me a compliment throughout. He did not do a thing to disturb me except to pay close attention to what I was doing and saying. I got off with the first case without any trouble. I had no notes. I was thoroughly accustomed to chalk and blackboard in teaching, and I made freehand drawings to clarify the roentgenogram and got along very well with this case for a few minutes. As I started to walk toward the left side, I experienced a phenomenon that was new to me. In spite of myself, I felt my vision being pulled toward Dr. Halsted, and when my eyes met his, I was through with

Case 1. I could not say another word. I dismissed that case and nodded for Case 2, turned toward the right and knew at once that I had to avoid looking at Dr. Halsted until I was through with each case. I had enough cases to fill the hour, and I did not look at Dr. Halsted until I had finished each case. I got through, I believe, without anyone noticing that I was unusually disturbed, or realizing that Dr. Halsted stopped me cold when I looked at him. The unusual thing about the clinic was that often afterward I would meet a member of that class who would say something pleasant about that particular clinic. When the clinic was over, Dr. Halsted came over and was pleasant in manner and very courteous, and my temporary insanity immediately disappeared. One explanation may have been that I had watched Dr. Halsted operate often and I always felt that there was an unusual amount of tension among the men who were assisting him.

Later he sent me, with New Year Greetings, bound copies of two of his famous surgical reports: "Ligations of the Left Subclavian Artery in the First Portion," and the "Operative Story of Goiter." He had written a friendly greeting on the cover of each of them.

The last remembrance I have of Dr. Halsted was an invitation to dinner at his home to meet a famous Dutch surgeon who shall be nameless. Dr. Halsted's dress and manner were in perfect taste. I noticed that there were no creases in the table cloth. He had apparently arranged the menu himself. Mrs. Halsted was not at the table. The thing that makes me remember the details of that dinner was the fact that almost every dish was maize prepared in a different manner, and the foreign guest's uncertainty as to what they were. Dr. Halsted was the perfect host and the service was perfect. He talked more than I expected. The North European surgeon was game and equal to this meal that was arranged for his curiosity and edification. He undertook each strange dish with equanimity and followed just a little after the others in eating his dinner. I could not make up my mind as to the extent of his uncertainty, because I was myself not always sure of the next dish. But the dinner and company were good, and Dr. Halsted showed a surprising knowledge of the many ways in which corn may be eaten. Not a thing went amiss. I got through almost as well as the special guest for whom the menu was arranged. The only thing I was very careful about was the very black and thick coffee. I had heard other men speak of staying awake the entire night after a cup of Dr. Halsted's coffee, but it was a pleasant evening because Dr. Halsted made it so.

# A PEDIATRICIAN'S CHANCE RECOLLECTIONS OF DR. HALSTED ·

EDWARDS A. PARK, M.D., BALTIMORE, MD.

ALTHOUGH a great admirer of Dr. Halsted, I came to know him more particularly because of his great interest in the studies in which Dr. McClure and I were engaged on the thymus gland. One of the well-known German surgeons (Garre) ascribed the toxic symptoms of goiter to the abnormal activity of the thymus gland and for a time Dr. Halsted resected the thymus gland in addition to the thyroid in the treatment of toxic cases. I think that Dr. McClure's and my work were instrumental in convincing Dr. Halsted that the thymus gland had nothing to do with the toxic symptoms of Grave's disease.

Samuel Bushnell was Dr. Halsted's roommate at Yale for three years. After graduation from Yale, Bushnell studied for the ministry and was for years the pastor of the Central Congregational Church in New Haven, Connecticut, where he was greatly respected. Rev. Samuel Bushnell remained throughout his life one of Dr. Halsted's closest friends. When Dr. William G. MacCallum was engaged in writing his biography of Dr. Halsted, I volunteered to call in his behalf on Mr. Bushnell and also on Dr. Fred Shattuck, who remembered Dr. Halsted during his period of drug addiction when he was a patient at the Butler Hospital, Providence, Rhode Island. I did interview both Mr. Bushnell and Dr. Shattuck but Dr. MacCallum never made use of the material. However, notes on my interview with Mr. Bushnell, dated Feb. 23, 1927, were found among Dr. MacCallum's papers, but the notes of the interview with Dr. Shattuck seem to have disappeared entirely. I shall begin by reading relevant parts of the record of my interview with Mr. Bushnell. There is a good deal of repetition but perhaps this lends a vividness to what he had to say.

BUSHNELL: I was a New Haven boy and lived at home. In my senior year I lived in Connecticut Hall in room 41 right over Dean Mendell's office, then in Durfee. Halsted slept in a little alcove in our room. At that time the faculty made up their minds how the students stood scholastically. Halsted was in the second division. At one time he made up his mind that he would dig and get above the second division and worked very hard, but at the end of the term when the marks were given, he found that he had remained in the same place, and then "quit." From that time on he made no effort for scholarship, but had great ability.

PARK: Was he a remarkable athlete?

BUSHNELL: Fifty years ago he was actually the leader of football. But it was not then the thing it is now. We had three more remarkable men than Halsted; they were Nevin, Maxwell, and Bonds. He was inferior to them but was excellent in football. He was agile and intelligent in a general capacity. The most notable game in which he figured was played Dec. 6, 1873, with an eleven made up of graduates of Eton, England, which Yale won 2-1, Captain Halsted making the winning goal. He rowed on the class crew in his sophomore year and was a member of the class nine and captain of the varsity football team in his senior year. He never tried really to be an athlete—he took it as a past-

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time. Though rather slight in size, he was exceptionally strong and could handle a 100 pound dumbbell easily and took part in a gymnastic exhibition in behalf of the Yale Navy, March 1, 1873. He weighed 160 pounds; he had good muscle in his arms, a big thumb, and outstanding ears. He was blond and had a mustache. He was in perfect health and was in all respects a normal man.

Halsted later became very much interested in certain sports and for a time went to prizefights. There was no such thing as prizefighting when he was in college. He was a boxer; he knocked me down flat once. He was very quick, alert, and strong. He had great strength in his arms and legs. Sparring had not yet come in; it was just coming in then. I have not boxed since he knocked me down. Halsted did not shoot and he was not a fisherman.

PARK: Did he show no particular interest in any subject?

BUSHNELL: No, he took the general course and had no more interest in any one department than in another.

PARK: It was not because he was coerced, was it? Did he rebel against conventions? Was it because he was supposed to do things that he did not do them?

BUSHNELL: I think he was rather cynical. He had a capacity for saying sharp things. He was cynical, in a way, but he did not consider the impression he might make on men in that way. This made him a little unpopular in some respects; it retarded him with some men; it was an expression of his mentality. He would say things that were cutting.

PARK: Was he sarcastic with his teachers?

BUSHNELL: No, he was respectful to his teachers always.

PARK: Did he show his fastidiousness then?

BUSHNELL: Yes, to a certain degree. He always dressed in good taste and was very careful about his dress. He was always one of the best-dressed men in his class. He had plenty of means but did not make any great show of it. He did not spend any great amount of money in college. He had a tailor make him a suit from bed ticking and wore it at college for two days. He induced one other friend, Frank Jenkins, to wear a similar suit. Halsted was the ringleader but did not have the courage to carry out his idea alone. Jenkins had the nerve to carry it through.

PARK: Was he fastidious about his food?

BUSHNELL: He had been at Andover and accepted anything.

PARK: Did he ever have escapades?

BUSHNELL: He did not drink; he had too much poise and common sense. He reacted against his father's type of Presbyterianism. He was religious but took no further stand. He never was brought before the faculty for anything. He had a great deal of self-respect.

PARK: What were his parents like?

BUSHNELL: He had a charming mother. She died of cancer while he was studying in New York. His father was William M. Halsted, of Halsted Haines and Company, New York, wholesale dry goods. His father was rather reserved and stern. He was not as easily accessible as most fathers.

PARK: Did he get his tastes from his mother?

BUSHNELL: His father was a very able financier in New York. His mother

was a very gentle creature. His ability probably came from his father. The Halsteds were connected with the Haines, good American stock, but no one in the family was exceptionally intellectual. It was a very old family. He was in a way a sort of sport. He had a brother Dick, and a sister Minnie who married Dr. Vanderpool. Bertha, a younger sister, married a Mr. Terry. Halsted possessed the brains of the family, undoubtedly.

PARK: Have you a photograph of him in those days?

BUSHNELL: He was very shy about having his picture taken. He avoided having pictures taken if he possibly could.

PARK: What made him choose medicine? Did he know when he was in college that he would study medicine later?

BUSHNELL: I think I know what started that man—his failure to get into a senior society. The only reason that he did not was because elections were made by the class above him and they did not know him. There were 124 men in his class who finally received degrees. There was only one place he wanted to go and that was Skull and Bones. I had a great affection for Halsted and we had been together for three years then and I said I would refuse my election if he did not get one and he said he would cut my friendship if I did that. He would not stand for it and added that every man must go on his own merits, as recognized by the class above.

"If you get an election, you take it; if I get an election, I shall take it. I shall expect you to do the same by me," is what Halsted said.

He would not be dragged in by me—and he was very much disappointed.

His father said to me, "Why didn't you get him into Skull and Bones? You made it."

His father was disappointed. Halsted swallowed his disappointment like a man and he resolved then to show his contemporaries and classmates that he would do something. Either because of the fact that he was conscience-stricken that he had not done much in his four years in college—he had not been a scholar—or a sense of futility stirred him to show his classmates that he could do something. The reaction came when he graduated and he made the finest record after leaving college of us all. He was pre-eminent among all us men and was of greater personal service than anyone else in his class. As soon as he went into medicine in New York, he worked like thunder and at the end of two years he applied for a position at Bellevue. One of the professors refused to consider him but another said that there must be some reason why he should present himself for examination with so short a period of study behind him and should be given a chance. He passed the examination better than anyone. He had enough money to buy cadavers, which all his classmates did not have, and spent all his time studying dissection. At that time men were studying to prepare for medical practice and not for surgery and did not need cadavers. Halsted bought all cadavers not desired by the men studying to be physicians. He had fully decided then to become a surgeon. I knew before the end of his senior year that he was heading for the College of Physicians and Surgeons. After graduation he went right down there.

PARK: Was Dr. Halsted a pleasant roommate?

BUSHNELL: Very delightful.

PARK: Was he fond of books?

BUSHNELL: Yes, he read a great deal, more than many young men in college. He did not have much modern literature in his library; he read classical fiction.

PARK: Did he have literary tastes?

BUSHNELL: Yes, and he had good tastes and he had a keen mind. He read French fluently. We had French and German in college. Whatever he did, he did well and concentrated on. He would learn a play four times as quickly as I. He took parts in plays, usually the parts of men, mostly humorous parts, acted well and was quick. He would learn very quickly. If he had only applied himself scholastically he could have had many honors. He had ability to spare.

PARK: Was he interested in social life?

BUSHNELL: There were a few of us who had sisters here in New Haven and he had access to families but, while he liked girls, he did not go in for social things. He danced well. He was very fond of dogs—he had them in Baltimore. He was interested in them and often dissected them. He had no horses.

PARK: How did your friendship begin?

BUSHNELL: I cannot tell how we came together. It was a natural affinity. He was attracted to me. We were great friends through life. If I had not gone into the ministry I would have gone into medicine with him.

PARK: Was Dr. Welch two years ahead of Halsted?

BUSHNELL: He was four years ahead. He was his greatest friend after college. He chose him for the place at Hopkins. He stuck to Halsted when he tried out cocaine and lost control.

PARK: Did Halsted have courses in science with Silliman or Dana?

BUSHNELL: Yes, he had Dana.

Dr. Fred Shattuck, whom I interviewed after Mr. Bushnell, was, of course, the distinguished professor of medicine at Harvard University. I cannot recall exactly what the connection of Dr. Shattuck, as a young man, with Dr. Halsted was but, though the record of my interview cannot be found, the following recollection comes to mind. Dr. Sawyer was the head of the Butler Hospital. He died in December, 1885, when Dr. Halsted was a patient there. Dr. Shattuck said that as he, Dr. Folsom, who later became a well-known psychiatrist, and Dr. Halsted were standing together at Dr. Sawyer's deathbed, Dr. Sawyer's last words were an appeal to Dr. Halsted to abandon his drug addiction.

It is interesting that Dr. Arthur H. Ruggles in his historical account of the Butler Hospital entitled, "A Century of Butler Hospital" stated that the special contribution of Dr. Sawyer was "the first instance of the use of surgical consultants for operative procedures to improve the physical condition of the patients." One cannot help wondering whether it was not the influence of Dr. Halsted which caused Dr. Sawyer to introduce this innovation.

Since Dr. Halsted's drug addiction has been referred to, I wish to express an opinion about it. Apparently there are those who say that Dr. Halsted completely recovered from his drug addiction and those who insist that he never succeeded in recovering. The latter group, so far as I know, base their opinions on circumstantial evidence, in some instances of the vaguest kind. Dr. Richard Follis, Jr., one time asked his father for his opinion and Dr. Follis, Sr., who was

one of Dr. Halsted's special favorites, replied that his contact with Dr. Halsted on a European tour was of so constant and intimate a nature that it would have been impossible for Dr. Halsted to have administered cocaine or morphine to himself without his knowing. I should like to call attention to another consideration. So far as I know, addiction to cocaine or morphine never remains a constant over the years but moves either in one direction or another. Either it is given up or the habit progresses, as in the case of De Quincey. It would seem to me impossible that Dr. Halsted could have pursued the absolutely even tenor of his way year after year until he was 70 years old if his drug addiction had continued. The question whether or not Dr. Halsted recovered is an entirely academic one and has only an academic interest, for his life to the very end was one of uninterrupted glory.

I now wish to give a few personal recollections of Dr. Halsted. When you walked behind him, you were conscious that his elbows stuck out from his sides. Dr. MacCallum called attention to this as indicative of the large development of his muscles. Dr. Halsted's figure seemed so slight that it was difficult to imagine him as possessing unusual strength.

I was present one time at an operation by Dr. Roy McClure when he was resident surgeon. McClure thought that the wound was infected and was engaged in using catgut sutures when Dr. Halsted entered. As Dr. Halsted saw what McClure was doing, he became very red in the face, turned on his heel and left the operating room. He never said one single word, but his behavior was daggers drawn. I am not sure that McClure was not condemned in Dr. Halsted's mind from that moment forth.

My daughter, who was about 6 years old, managed to cut the extensor tendon of the left index finger between the middle and distal joints so that she could no longer extend the distal phalanx. I took her to Dr. George Heuer and, while he was examining her, Dr. Halsted happened in. Heuer asked Dr. Halsted to look at the finger. Dr. Halsted became at once greatly interested and asked Heuer to make dissections on 2 or 3 cadavers before operating so that he would have exact anatomic knowledge, and also insisted that Dr. Richard Follis be called in consultation. This indicates the care which Dr. Halsted insisted on in the approach to even a common problem. Dr. Halsted was utterly charming in his approach to my little daughter, winning her confidence at the outset. He was extraordinarily kind at that time, and I could give other illustrations.

Every quality which Dr. Halsted exhibited was on the same elevated plane. He was an aristocrat in the finest sense of the word in that every action, thought, and expression possessed the same rare quality. Nothing ordinary or mean ever came from him. This excellence was true in regard to his wit which had a rapier-like quality. As a boy in Phillips Acadamy he was forced together with all the other boys to hear my grandfather preach.

He remarked to my sister once, "He preached well, but an hour."

Dr. Baetjer of the X-ray Department told me that once when he was talking to Dr. Halsted in the hallway Dr. Bloodgood passed by. Dr. Baetjer said that he noted that Dr. Halsted kept turning away from him to follow with his eyes the retreating figure of Dr. Bloodgood.

Finally he observed, "There goes Bloodgood. He can draw more general conclusions from a single instance than anybody I ever knew."

At a medical meeting at which the speaker had presented a subject in biochemistry difficult for even a trained chemist to follow, Dr. Halsted, who was sitting in the front row, was called on to discuss the paper.

He said merely, "The speaker has talked over my head to the students above," and sat down.

Dr. Halsted remarked to me one time, "At medical meetings the best-looking men are the internists; the worst-looking men are the dermatologists and the surgeons come in somewhere between."

When Dr. Welch resigned from Bellevue Hospital in order to become Professor of Pathology at Johns Hopkins, the Committee at Bellevue, feeling it necessary to replace Dr. Welch with a man of great distinction, invited Dr. Lubarsch, the well-known German pathologist, to visit Bellevue with a view to his consideration as Dr. Welch's successor. Dr. Halsted told me that at the farewell dinner to Dr. Welch, he was seated opposite Dr. Lubarsch.

They were eating corn on the cob when Dr. Lubarsch remarked to Dr. Halsted, "I wish I had a picture of you eating corn on the cob."

Dr. Halsted said to me that he knew if he talked very fast Dr. Lubarsch could not understand.

So he said, "I wish I had a picture of you, Dr. Lubarsch, combing your beard with your fork."

Dr. Lubarsch had a long black beard. Everybody laughed and Dr. Lubarsch was never able to find out what Dr. Halsted had said.

Dr. Halsted and Dr. Welch used to play tricks on each other and I was most pleased to find that the inherent greatness in these men had not removed all boyish qualities. I was told that at a dinner at the Maryland Club Dr. Welch had placed under Dr. Halsted's plate a rubber bladder connected by a rubber tube with a bulb and Dr. Welch took great delight at the mystification of Dr. Halsted when his dinner plate from time to time danced up and down.

Dr. Halsted himself told me that a German professor one time sent him for Christmas an umbrella accompanied by his card. Not caring particularly for the umbrella or the German professor, Dr. Halsted sent the umbrella, with the card attached, to Dr. Welch on the day before Christmas. Dr. Welch concluded that Dr. Halsted had sent the umbrella and wrote Dr. Halsted a letter of thanks in Latin. Dr. Halsted was determined "to find some mistake in Welch's Latin" and so got out his grammar and his lexicon but was unable to discover any. Major Venable, who lived at the Maryland Club and was a great friend of both Dr. Halsted and Dr. Welch, was a Latin scholar and consequently Dr. Halsted submitted Dr. Welch's letter to Major Venable. Major Venable reported that Welch had got his cases, genders, numbers, moods and tenses all right but it was doggerel Latin.

When Dr. Halsted reported to Dr. Welch what Major Venable had said, Dr. Welch laughed and remarked, "Yes, Sallust* wrote doggerel Latin."

*My recollection is that Dr. Halsted named Sallust as the author. But the author certainly was not Sallust. I have never been able to discover who the author was unless it could have been Martial, who wrote a letter of presentation of a sunshade to a friend. Martial's letter could have been altered so as to apply to Dr. Halsted's gift of an umbrella.

He had copied the letter from Sallust.

I have heard Dr. Finney tell this story about Dr. Halsted: An elderly maiden lady was sent as a patient to Dr. Halsted on account of an abdominal tumor. When Dr. Halsted was unable to make a diagnosis, the family asked Dr. Finney to see the patient. Dr. Finney discovered that the woman had been taking tansy tea and putting two and two together made the diagnosis of an ectopic pregnancy. The family asked Dr. Finney to operate and Dr. Halsted assisted. It turned out that the condition was an ectopic pregnancy.

After the operation Dr. Halsted remarked, "It is better to make no diagnosis than the correct diagnosis without a reason and, besides, Finney has a superior knowledge of the world."

How extraordinary it was that there should have been in the Medical School a Professor of Surgery with the qualities of mind and habits of Dr. Halsted, such as one associates with the great thinkers of the world, philosophers like Descartes or mathematicians like Einstein, rather than men of action. Certainly Dr. Halsted realized the value of thought and a large part of his day was given over to thought. I believe that his habit of apportioning so much of his time to thought was the secret to his accomplishments as well as perhaps his most distinguishing characteristic.

In conclusion, there are two kinds of teachers, *conscious* and *unconscious*. The conscious teacher is primarily aware that he is teaching. His aim and what he regards as his business is to transfer facts or ideas from his mind into the minds of his pupils with the least expenditure of energy on both sides. The unconscious teacher is totally unaware that he is teaching. His teaching is by example. When I look back on my life, it seems to me that the teachers who gave me the most and to whom I feel the most grateful were not those who made passage of knowledge easiest, but those who stimulated me in one way or another to strive to be like them. Those unconscious teachers of whom I speak possessed greatness, in some instances of an intellectual, but often of a moral, kind. It was shown, perhaps, by demands for clearness and accuracy of thinking and expression, by liberality and generosity, by the elevation of their standards and ideals, or by the breadth of their outlook and perhaps, also, by their unconsciousness of themselves. Dr. Halsted was a magnificent example of what I mean by an unconscious teacher; his influence pervaded the medical school and hospital and was felt by me in the Harriet Lane Home for Invalid Children. We who knew him longed to be like him, as a young would-be artist might long to be like a great artist, the work of whose mind and hands, wherever directed, seemed to have about it a certain perfection. Dr. Halsted's spirit and standards automatically became the spirit and standards of his younger associates so that one might say that those serving under him became molded in his image and went forth as missionaries of his gospel. This celebration of the centenary of Dr. Halsted's birth is as much a tribute to the strength of his unconscious pervasive influence as it is a memorial to his measurable accomplishments.

# PROFESSOR HALSTED AS I KNEW HIM IN THE HUNTERIAN LABORATORY FOR EXPERIMENTAL SURGERY

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MY FIRST contact with Dr. Halsted was in the weekly surgical clinics which often fell short of the expectations of his student listeners because of their inability to comprehend the wide implications with which he sought to arouse their scientific interest.

My personal contact with the Professor came in the fall of 1920 when, after finishing medical school, I found I had been assigned to the Hunterian Laboratory. The Professor asked me what I wished to do and, as I had no problem, he told me of a nurse who had nearly died from *Escherichia coli* infection after resection of the descending colon adherent to a uterine tumor. He felt that a simple aseptic type of anastomosis was necessary and he had devised the cork-tipped, spear-pointed knife attached to a flexible tube that could be introduced by rectum and guided to the bulkhead of an aseptic end-to-end anastomosis. When the surgeon's fingers grasped the knife from above, the cork could be freed and the knife pushed through to cut the purse-string sutures, thus opening the lumen aseptically.

In his conversation that first day he asked me if I had read Billroth and Cohnheim. That night in the library I found English translations in the New Sydenham Society series that permitted me to read these fascinating books. I not only read them but secured several copies of my own which I have continued to advise the men in the surgical research laboratory to read, as I did then.

The cause of the swelling of the arm after radical mastectomy, which was rare after primary skin grafting, but seemed to develop when a wound sloughed or infection occurred, interested the Professor as being due probably to lymph stasis. To produce lymphedema, he had suggested to the late Dr. Mont Reid, then resident surgeon, that he do a mid-thigh amputation, leaving the major vessels and bone, but dividing muscles, veins, and lymphatics. I continued the experiment with Dr. C. Y. Bidgood and found that the technique could be improved. By carefully approximating the fascia of the muscles, then the subcutaneous tissue and the skin, we soon ascertained exactly when the veins, arteries, and lymphatics regenerated across the site of amputation. Working with Dr. Reid, we were able, in one animal, successfully to divide the femur in a zigzag fashion, wire it together, then divide and resuture the femoral artery and vein and the sciatic nerve, as well as all other tissues.

This replantation study was presented to the National Academy of Science and also at the 1922 meeting of the American Surgical Association in Washington. Dr. Halsted took me to this latter meeting and pointed out some of the distinguished surgeons in the Association, a memorable experience. His

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comment that in his opinion it was the most worth-while surgical association in America made a lasting impression. I also remember the big fresh strawberries, so expensive in May, which he ordered in the Union Station before the train left.

I wonder what has become of a colored print of Cosmas and Damian, given to the Professor by some friend, in which the lower extremity from a dark-skinned person had been sutured to the amputated thigh of a white man!

In his clinics, Dr. Halsted had emphasized that the more distal from the heart an artery is ligated, the higher is the percentage of resultant gangrene. He told of earlier experiments by the late Dr. Philip Gilman in which ligation of the abdominal aorta had produced no gangrene, but in older dogs, weakness and even paralysis of the hind limbs had been noted the next day. He was interested in Luigi Porta's illustrations of 1845 that showed a mass of unnamed vessels between the ligated and divided ends of the lower abdominal aorta. Dr. Halsted thought these unnamed vessels might be vasa vasorum, so another experiment was started in which young dogs were used so as not to incur again the paralysis that he and Gilman had found. We learned, after dividing the abdominal aorta in these young dogs, that the great collateral bed between the ends of the aorta, depicted in Porta's illustrations, was due to the dilatation of the lumbar segmental arteries.

Dr. Halsted, after one of these experiments, would telephone in the afternoon to inquire about the animal, and although I reported that the animal was out of the anesthetic and up and about, wagging his tail, he would insist that the dog might be suffering and tell me to give him up to a quarter of a grain of morphine. On the wards at that time, the most that we dared give a patient was an eighth of a grain.

The days were gloriously busy in assisting Dr. Halsted or in helping the late Dr. Heuer in his pneumonectomy experiments, or in performing hypophysectomies in puppies for the late Dr. Dandy. The studies made of the sacrificed animals in these various problems were so engrossing that day and darkness often merged as one.

When Dr. Halsted left for the summer, there was so much more to do that I asked him for permission to stay on in the Hunterian for another year. He explained that I must learn clinical surgery, that from contacts with patients come the experiments in the laboratory. However, during my next year in the hospital, he would personally come into the operating room, two or three times a week, requesting the surgeon that I be permitted to leave the team. Then we would go over to the laboratory to continue the experiments begun the year before. Some of the problems that he started me on then were not completed until nearly ten years later.

The principles of surgery so constantly emphasized by him; namely, absolute asepsis, complete hemostasis, gentle handling of tissues, and careful approximation of tissues were applied as carefully in the experimental laboratory as in the operating room. There was no reason to hurry and in making an end-to-end colonic anastomosis, his delicate and deliberate placing of

each silk stitch so as to obtain a bite of submucosa, and his comments as to whether or not the needle might have penetrated into the mucosa, were recorded in the protocols. Months later, when the specimens were cleared by the Spalteholtz method and studied, the fate of these sutures, so carefully placed and so meticulously recorded, became known. Other specimens obtained from anastomoses performed with continuous catgut were cleared and compared with these made by Dr. Halsted. It was his conclusion that the healing was influenced less by the suture material than by the skill of the surgeon.

In the laboratory we learned to know the Professor as a perfect gentleman, imperturbably calm even when his match ignited an ether cone with which a dog was being anesthetized. I rushed to the sink with the cone but, unmoved, he quietly reminded me that my efforts were unnecessary, that the dog was dead, and that there would have been no fire. His manner was quiet, always, and deliberate. He was a man who made suggestions but never gave orders. Although he was supposed to have piercing steellike eyes, to me they were intelligently sympathetic, except on one occasion, when he became aware that he was being told a falsehood.

His expression changed immediately and as we walked on he said in his quiet tone, "Why should he have lied to me?"

It was to him a personal violation of trust.

He showed a keen interest in ordinary affairs, as evidenced by his fascination with the dexterity of the operator of the steam shovel when the old pathology laboratory was being replaced by the present building.

When he and Mrs. Halsted left for the south in the summer of 1922, he had many problems in mind for which he was planning experiments to be done that fall, but his death left them and us without his quiet, keen guidance.

# THE COMING OF FULL TIME TO THE HOPKINS AND HALSTED'S ATTITUDE TOWARD THE PLAN

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THE time at my disposal this morning does not permit a detailed recital of the events which led to the introduction in 1913 of the so-called full-time system into the clinical departments of the School of Medicine and Hospital. I shall have to content myself with a very brief statement concerning them.

Many of our own graduates were extremely skeptical of the wisdom of the move when it was made, and some were even bitterly opposed to it. Many of those who did not like the idea were accustomed to regard it as a move which was foisted upon the Medical Faculty by outside interests, in particular certain officials of the Rockefeller philanthropic organizations, but, after surveying all the material that I have been able to get hold of, I am convinced that such was not the case.

To me, at any rate, the evidence is overwhelming that the idea originated with Carl Ludwig in Germany and was first advocated in this environment by Dr. Mall who got it from Ludwig, under whom Mall had studied. Both Dr. Barker and Dr. Howell had also advocated the full-time system for the clinical departments in public addresses prior to 1910. The idea certainly did not originate with Abraham Flexner, as Mr. Flexner himself has said,* although he did have much to do with getting the move under way here at Hopkins.

You are all familiar with the epoch-making survey of medical education by Abraham Flexner in 1910. Please remember that he made his survey while he was a member of the staff of the Carnegie Foundation for the Advancement of Teaching, and at the request of the Foundation's president, Henry S. Pritchett. At the time he undertook the investigation Flexner had not yet become identified with any of the Rockefeller organizations.

When Flexner's report appeared it at once attracted the attention of Mr. Frederick T. Gates, who was then the confidential adviser of John D. Rockefeller, Sr., in matters of philanthropy. Mr. Gates sent for Mr. Flexner and asked him what he (that is, Flexner) would do if he had a million dollars with which to make a start in reorganizing medical education. Flexner replied, "I should give it to Dr. Welch," and then went on to give the reasons for his statement. Out of that meeting grew a visit by Flexner to Baltimore in an endeavor to determine to what uses the income on a million dollars of new endowment could be put in the Medical School and Hospital. According to Flexner, when the subject was first broached at a small dinner at the Maryland Club given by Dr. Welch, Mall was the first to speak up and suggested that it should be used to place the leading clinical departments on a full-time basis. Halsted was present at that dinner but unfortunately we do not know what position he took at that time, although

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*Flexner, Abraham: *I Remember,* New York, 1940, Simon & Schuster, Inc.

Dr. Welch has said that he was in complete sympathy with the full-time idea. We do know that he voted for the proposal when it came before the governing board of the Medical School.

Fortunately for our purpose Dr. Halsted left among his papers some notes in his own handwriting concerning the full-time proposal and, after it was approved by the authorities, addressed to the Trustees of the Hospital and of the University a memorandum on how the plan would affect the Surgical Service of the Hospital. Since this material has never been made public so far as I am aware, it seems proper to acquaint you with it at this time.

I shall not read all of his notes but have selected one (No. 12107) that seems to me may be of particular interest to you. It was written in pencil on his own private stationery.

DOCUMENT 12107

1. The clinical men tend to deteriorate in certain directions, & the laboratory men are prevented from developing in others.
2. The world over the laboratory men are of a higher order than the clinicians; & among the clinicians the physician outranks the surgeon & the surgeon outranks the gynecologist.
3. As a rule perhaps, the more intellectual men & those with the highest ideals select the laboratory branches, but there are many whose careers are determined by slight influences, & others who if they had as young men appreciated what is meant to vow themselves to poverty would not have done so. Some think or realize very early that their relations with their fellows are not so easy as to encourage them to risk the attainment of a success which is in great measure dependent upon personal relations.

   The reward for the greater talents & higher achievements should be advance in position & not increase in wealth.

   Public opinion viz. what is expected of a man (has) great influence & hence a man would be held in check in his greed for money.

   I am free from prejudice in trying to take position in this matter, because it would now make, so far as my income is concerned, very little difference to me whether it (my income) was limited to a $10,000 salary or not. The assurance of such an income, which is perhaps a few thousand dollars less than what I am at present making, would quite compensate for the uncertainty of making so much in the future.

   . . . . .

   The ideal simple life is that which is not enforced.
   The simple life is not ideal unless arrived at by voluntary renouncement of luxury.

Another document (No. 12103) deals with the reorganization of the surgical dispensary, a third (No. 12108) is an amplification of No. 12107. And then, finally, there is the memorandum addressed to the Trustees of the Hospital and University which I would like to read in full. This memorandum was sent to Dr. Hurd with a covering letter dated Dec. 16, 1913.

TO THE TRUSTEES OF THE
JOHNS HOPKINS HOSPITAL AND UNIVERSITY

We are committed to the policy of the All Time System and are making an experiment which will be watched with keenest interest not only in this country but abroad, by the public as well as by the men who are devoting themselves to the cause of science and education. It would be a pity if those of us to whom the working out of this plan

is entrusted and who are convinced of the wisdom of the Trustees in endorsing it by their acceptance of the gift from the General Education Fund should not promptly apply themselves to the solution of some of the most important of the various problems which at the outset demand their immediate attention. For the Surgical Department the problem in general may be especially difficult unless provision is made at the Hospital for the accommodation of a much greater number of patients. To make this clear it probably is not necessary to outline in any detail what is contemplated.

We must attract to the service men of unusual ability and promise of exceptional talent for the art as well as for the science of surgery; but we may not hope to do so unless we can offer them opportunities for the practice of their art as well as facilities for research.

We should fail utterly as surgeons were our staff composed chiefly of laboratory workers who could not be given patients for treatment.

The general surgical service is at present so small that, although I reserve for operation by myself perhaps less than one-tenth of the ward patients, the house surgeon finds it impossible without serious sacrifice to the development of himself and perhaps also of the welfare of the patients to assign operations to the other members of the staff, to transfer to them the opportunities to which he is entitled and which for the sake of the patients and reputation of the Hospital and Medical School it is absolutely essential should be afforded him.

For the education of a surgeon in his art many years of active operative work are essential. For the training even of our present staff and for the proper instruction of our medical students the number of surgical patients is inadequate.

This limited accommodation for patients handicaps the surgical perhaps even more than the medical departments, although I am not sure that this is so. The majority of the patients occupying surgical beds cease to be of particular interest after operation, for their convalescence is uneventful. Even the first dressing which only a few years ago was made with a little apprehension, the verdict as to healing being eagerly awaited, is now an event in which the members of the staff take little interest so accustomed have they become to the healing of wounds by first intention.

Men of the type desired for positions on our staff must have opportunities not only for research but for rapidly increasing their technical equipment for their vocation.

Furthermore the problems which are to engage the supreme efforts of our young men should be chiefly those which have a surgical bearing. Such problems are suggested in the course of work in the wards and operating rooms and particularly by the care and treatment of cases for which the surgeon feels a personal responsibility.

In a word, for the success of the Surgical Department on its new basis it is necessary that the clinical facilities be greatly increased, for, if not, we shall fail to attract to this service men of the type not only ardently desired but essential to the carrying out of the plan to which we have pledged ourselves to adhere.

I am sure that you will all agree with me that these words of Dr. Halsted are as true now as they were when they were written by him nearly forty years ago.

# THE STORY OF THE HUNTERIAN LABORATORY

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THE FULL story of the Hunterian Laboratory is a long and varied one. Of necessity this must be true, since during its forty-seven years of existence there have been three professors of surgery directing its activities, and more than one hundred investigators carrying on their researches in it. A laboratory is not merely a place in which to work. It is not just a collection of apparatus, but is, in essence, an atmosphere created by those who work within its walls. To borrow from Milton, one might say that laboratories, like books, are not altogether dead things, but do contain a potency of life in them to be as active as the soul whose progeny they are. This is indeed true of the Hunterian Laboratory, which, during its early years, portrayed with the precision of an etching the spirit of the man we honor today.

To compile a list of every surgeon, fellow, and student who worked in the laboratory would give us no true picture of the place; nor would a tabulation of all the publications emanating from this laboratory give us an accurate measuring of its influence. Just as one does not acquire a true understanding of history by listing names and dates, but does so by discovering those larger tendencies, those climates of opinion, those conditions that find their expression in recorded events; so in telling the story of the Hunterian Laboratory one must discover, first, the circumstances and conditions under which it came into being, and second, the philosophy or atmosphere which characterized and colored its activities.

The erection of the Hunterian Laboratory grew out of the need for space for teaching third year students.

In a letter to Dr. Welch, Dr. Halsted wrote: "I should be sorry to have it forgotten that I initiated the operative course on animals. Courses in experimental surgery were given to the first class in their third year (1895). One of the inducements offered to Cushing to return to us was the transference of these courses to him."

The first classes in operative surgery were held in the corner room on the first floor of the Anatomy Building, which later became Dr. Sabin's laboratory. Dr. Mall needed this room, and in 1903 Dr. Cushing requested the trustees to erect a small, inexpensive building. Had not the disastrous fire in February, 1904, caused great financial loss to the University, it is likely that this request would have been granted. When denied, Cushing with characteristic zeal solicited the aid of friends, and was promised five thousand dollars, but the trustees declined to accept the gift stating that the sum was inadequate to put up a building in conformity with the other buildings on the Medical School lot. Late in 1904, according to Cushing, MacCallum entered into a conspiracy with him and made another appeal to the trustees. This appeal was backed up by

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the medical students and brought results. The trustees voted fifteen thousand dollars for the erection of the building which was completed in the summer of 1905. Half of the space was allocated to the Department of Pathology and the other half to Surgery. Discussion arose as to the name for the laboratory. Cushing was in favor of calling it after Magendie, but this name was anathema to the antivivisectionists in Baltimore. Dr. Welch suggested the use of John Hunter's name, hence the Hunterian Laboratory for Experimental Medicine. This was not a bad solution, for it mystified many good Baltimoreans who thought that the term had some reference to pointers, retrievers, and setters.

The second phase of our enquiry concerns itself with the philosophy, the atmosphere, which characterized and colored all the activities in the new laboratory. These activities must be divided into three categories: the teaching of students, veterinary surgery, research. Concerning the teaching of students, Cushing stated that the novel feature in the course in operative surgery lay in the attempt to liken the exercise so far as possible to the actual performance of surgery as conducted on a patient in the hospital. The writing of a history, the keeping of an anesthesia chart, the writing of operative and postoperative notes, the performance of a complete post-mortem examination were integral parts of the exercise. This type of teaching surgical principles to students was as revolutionary as Osler's bedside instruction in medicine.

The second activity of the laboratory was the practice of veterinary surgery on a very high plane. For many years the laboratory was the haven of distracted dog lovers who brought their pets to it for treatment. The great interest in the natural diseases of canines is shown in the first three sets of papers that issued from the laboratory under the title *Comparative Surgery.* The observation of diseases of dogs led inevitably to efforts to reproduce these diseases in normal animals, and thence to application to human beings.

This sequence is beautifully illustrated by the opening sentence in a paper by Cushing and Branch: "We wish to mention briefly some attempts which have been made in the Hunterian Laboratory to reproduce chronic valvular lesions; to record certain cases of spontaneous valvular disease in the dog which have come under our observation, and finally to comment on the possibilities of future surgical measures in man directed toward the alleviation particularly of the lesion characterizing mitral stenosis."

Little did Cushing foresee in December, 1907, the attainment of this goal forty years later.

A careful study of the investigations carried on in the laboratory reveals three distinctive characteristics: First, the same excellence of performance demanded by Dr. Halsted in the operating room was required of the workers in the laboratory. Every detail of his meticulous surgical technique was applied to the operations on animals. Second, the majority of the problems studied were basic in nature. Third, a spirit of thoughtful inquiry pervaded the place.

No wonder then that the course in operative surgery was soon copied in other medical schools throughout the country. No wonder that the quality of

veterinary surgery as practiced by veterinarians was improved. No wonder that this small laboratory has served as a stimulus for the creation of similar laboratories throughout the country.

But the half has not been told. The influence of the laboratory on students, fellows, visitors in this country and abroad could not be measured. Actually, as laboratories go, the Hunterian is still young; it has barely reached its majority. It is cheering, however, to realize that the same attitudes which characterized the work in the early years continue. The spirit of inquiry, the interest in basic problems, and the excellence of performance are still dominant. It is reassuring to believe that the Hunterian Laboratory will continue to pass on the spirit of Halsted from one generation to another.

# COMMENTS ON HALSTED'S CONTRIBUTIONS TO ARTERIAL SURGERY WITH PARTICULAR REFERENCE TO PROGRESSIVE OCCLUSION OF THE LARGE ARTERIES BY MEANS OF THE ALUMINUM BAND

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IT IS a great privilege and pleasure to contribute my very small part to this program in observance and in honor of the one hundredth anniversary of the birth of Dr. Halsted. It affords me, furthermore, an opportunity to express my admiration of him and my appreciation and gratitude for all that my association with him has meant to me.

It so happened that after I graduated in 1909, and before I entered upon my clinical work, I was given a year's appointment to the old Hunterian Laboratory, in charge of the experimental surgical division. During this year I had the good fortune to assist Dr. Halsted in the furtherance of his experimental work on dogs on the application of aluminum bands to the larger arteries. I had the opportunity also of preparing histologic sections for the study of the walls of the large arteries constricted by the band. I realize keenly that my comments today fall far short of doing justice to the basic principles and fundamental concepts embodied in Dr. Halsted's many contributions on the subject of arterial surgery.

As stated so well by Dr. Matas, "The problems of the vascular system, especially of aneurism, had a great fascination for Dr. Halsted and on these he brought to bear all the resources of his keen, critical faculties and of his splendid training in anatomy, physiology and pathology."

There were twenty contributions on the surgery of the large arteries which appeared between May, 1892, and May, 1922, and are to be found in the Burket collection of Dr. Halsted's papers. Three concerned ligations, twelve or thirteen were on the experimental or clinical results obtained after the use of the constricting metal band, and the remaining ones were on the employment of fresh tissue bands, dilatation of an artery distal to a partially occluding band or cervical rib, arteriovenous fistulas, and periarterial sympathectomy.

The first contribution to surgery of the blood vessels was a report in May, 1892, on "Ligation of the First Portion of the Left Subclavian Artery and Excision of a Subclavio-axillary Aneurism."

Dr. Halsted stated, "This case is perhaps the only successful one of deligation of the first part of either subclavian artery, and the first one of complete extirpation of a subclavio-axillary aneurism." The brilliant result obtained in this case stimulated his interest in ligation of the large arteries and, before initiating any experimental work, he reviewed at least thirty cases of ligation of the common iliac recorded between 1880 and 1912. His object was to determine the mortality, the frequency of gangrene, the nature of the anastomotic circulation, and the ultimate result. There were fourteen fatalities attributable,

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in most instances, to causes other than ligation of the common iliac artery alone, a mortality rate of 46 per cent. In only two instances of gangrene was the ligation of the artery solely responsible for the gangrene, a percentage of 6.6 per cent, although gangrene had been reported in from 20 to 33 per cent of the cases. This high percentage, he believed, was due to the ligation of other important vessels concerned in the establishment of the anastomotic circulation. In many of the cases, wound infection was an additional complication.

Gangrene had never resulted from ligation of the abdominal aorta in the human subject nor did it occur in any of his dogs. He formulated the following important rule: that, in a general way, the larger the artery, or the nearer it is to the heart, the less the impairment of the circulation attending its ligation. He cautioned against ligations of the collateral branches of the common iliac. In excision of an aneurysm of the external iliac it was important to preserve the anastomotic channels of the epigastric and circumflex arteries to obviate the likelihood of gangrene which was commoner in this instance than after ligation of the common iliac.

Ligation of the common iliac cuts off the direct supply from the internal iliac and does not invariably cure the aneurysm. Accordingly, he hoped to find some means of progressively occluding a large artery to allow for the establishment of a competent collateral circulation and to avoid the complications attendant upon a too rapid decrease in the peripheral circulation. Thus, during the twenty-eight years between the publication of his first contribution on ligation of the subclavian artery in 1892 and his next paper on ligation in 1920, Dr. Halsted busied himself intensely with the development of the aluminum band and its application to the large arteries in experimental animals and in the human being for the treatment and cure of aneurysm.

In a preliminary report, March, 1905, he summarized the results of about ninety experiments performed upon the abdominal aorta and other large vessels of sixty-eight dogs. He felt the aorta might be successfully occluded in man if the operations and the occlusions were accomplished by gradual compression in stages. Metal bands of silver, at first, and later of aluminum were employed with the belief that at each operation the amount of constriction could be regulated to a nicety. The tightening of the band, the cylindric form being preserved with great care, was completed with the fingers.

He was afraid that the sharp edges of the bands would cut through the pulsating and constricted aorta, and, sure enough, on the twelfth day of the operation, a dog died from hemorrhage, the result of ulceration at the upper edge of the band. He feared that the procedure might be doomed but, no further cases of hemorrhage occurring, the experiments were resumed. Then, after about three months, he found to his chagrin that the wall of the aorta embraced by the band had, in almost every instance, atrophied, being reduced to a mere film in some cases. Notwithstanding this complication the experiments went on, in the hope that, with an improved technique, the walls of the arteries might retain their vitality. I remember how greatly impressed I was by the infinite care Dr. Halsted devoted to the details in the preparation of the band, all to obviate injury to the wall of the vessel with consequent thrombosis.

In these experiments thrombosis was not observed in a single case. It was shown by Reid that, when applied tightly enough to completely interrupt the circulation, the band caused atrophy and sometimes complete absorption of the aortic wall. Loosely applied bands on the aorta and other large arteries for months did not cause macroscopic injury to the walls of the artery. These results encouraged the belief that there was a place in surgery for the partially occluding band.

There followed soon after two instances in which it was used on the human subject. In the first case the band was applied to the left common carotid and in the second case a woman, asphyxiated to unconsciousness by an aneurysm of the arch of the aorta, died before the band could be applied. After these experiences Dr. Halsted felt that the smaller arteries, the ligation of which endangers merely the life of the limb, might prove as suitable for partial occlusion as the aorta, which had never been successfully ligated in man.

During the years 1904 to 1906 he conducted experiments on the aorta and other large arteries in 100 dogs and noted particularly that the pressure below the band was lowered, roughly in proportion to the amount of occlusion and that the return of pressure to approximately normal below the band was rapid. The incentive to this work was the desire to occlude safely the abdominal aorta or the common iliac arteries. Only one dog died of hemorrhage resulting from ulceration at the upper edge of the band.

In the clinical cases the band was particularly useful whenever it became necessary to test the effect of blocking an artery before permanently occluding it. If it was rolled too tightly and complications threatened, it might be removed at any time after operation. In a case of large popliteal aneurysm, the employment of the metal band to occlude the femoral artery enabled him to test the blood pressure during the gradual process of occlusion.

In a report in January, 1909, Dr. Halsted stated, "The aluminum band has now been successfully applied in man to the common carotid artery twelve times, and once each to the thoracic aorta, the abdominal aorta, the common iliac, the femoral, and the innominate arteries."

In 1909 he also reported three cases in which a partially occluding band had been applied to the thoracic aorta and the abdominal aorta.

In the first case (December, 1906) a band was applied to the thoracic aorta for treatment of a large aneurysm of the upper abdominal aorta. Convalescence was uneventful. Dr. Halsted was thus the first to occlude successfully the thoracic aorta for a huge abdominal aneurysm with a partially occluding aluminum band.

In the second case (December, 1906) an aluminum band was applied to the thoracic aorta in the hope of relieving the pain of a large upper abdominal aneurysm. Improvement followed. About three weeks later a band was applied to the abdominal aorta below the aneurysm. Recovery was uneventful in this case, also, but ten days later the patient died from intrathoracic rupture of the aneurysm.

In the third case (February, 1909) an aluminum band was applied to the abdominal aorta for aneurysm extending from its bifurcation to the renal ar-

teries. In spite of a great improvement in signs and symptoms, the aneurysm continued to grow and was wired by Dr. Finney. Finally there occurred complications of a suppurative nature which were secondary to operative infection introduced when Dr. Halsted was forced to remove his glove because of limited space in which to roll the band. The patient died. Dr. Halsted evidently was very chagrined because in a quotation he speaks of the suppuration as being "the first in the annals of the Johns Hopkins Hospital in a clean abdominal case."

In 1910 he reported two cases of probable cure of aneurysm—one subclavian and one external iliac—in the first case by partial obliteration of the subclavian, and in the second case of the common iliac artery. He had up to this time (June, 1909) no case to report of cure of an aortic aneurysm. In 1914 he reported a cure in a case of iliofemoral aneurysm in which partial occlusion of the left iliac artery by the band was employed (May, 1913) followed three weeks later by excision of the aneurysm. The results were no less than brilliant regarding the effect on the temperature, swelling, pain, loss of power and sensation in the left leg.

Ligation of the human abdominal aorta, always with fatal results, had been reported nineteen or twenty times. Furthermore, in one case in Europe in which Dr. Halsted operated, six weeks after the patient was discharged as cured, death occurred from hemorrhage as a result of aortic rupture at the the site of the band. These facts stimulated a search for something superior to the metal band in constricting the aorta. On one occasion I mentioned the possibility of using spiral strips of fascia lata which could be wound around the aorta. This procedure led to further experimentation but was finally abandoned, largely because of considerable absorption of the band with consequent loss of constriction of the aorta and complete restoration of the lumen after several months.

In evaluating the contributions of Dr. Halsted it can be said that the qualities of thoroughness, scientific honesty, accuracy, and keenness of observation, both in his experimental studies and in his approach to clinical patients, gave to his conclusions the greatest value. His treatment of aneurysm, a subject in which he retained the greatest interest throughout his professional life, was brilliantly successful in the case of the larger arteries other than the aorta, and this was true whether his treatment was by ligation or by the application of the metal band. The challenge of aortic aneurysm was, however, never satisfactorily met, largely because of the ever-present danger of hemorrhage, due largely to atrophy and erosion of the aortic wall produced by the metal band. The results were not good and after several trials it was concluded that it was unsafe to place metal bands upon the human aorta and it should not be attempted.

There was a gap of many years, following Dr. Halsted, during which little progress was made in the treatment of aneurysm. Finally, a number of surgeons, basing their procedures on the basic principles and concepts which Dr. Halsted had elaborated and employing new materials and techniques which were not available to him, succeeded in crowning his efforts to develop a method of safely and successfully occluding the diseased human aorta. These facts detract in no way from the importance of Dr. Halsted's contributions.

Thus Pearse, in 1940, reported his observations on dogs, noting that a profound fibrous tissue response occurred following the placement of cellophane about the aorta, and Owens, in 1942, noted a higher incidence of success when employing slingshot rubber bands upon the thoracic aortas of dogs. Cooper and co-workers, in 1949, reported on the gradual occlusion of the dog's aorta, using Polythene containing dicetyl phosphate beneath tantalum bands. Finally, within the past year, Blakemore reported a series of brilliant cures in seven out of eight cases in the human patient, in which he operated in one stage for aneurysms of the abdominal aorta by the employment of a method of constrictive partial occlusion of the abdominal aorta using the slingshot rubber band in conjunction with an underlying irritant plastic film. This constrictive occlusion was employed in conjunction with wiring and controlled electrothermic coagulation of aneurysms.

Time will not permit more than the merest mention of some other of Dr. Halsted's researches. He investigated the reasons why an artery dilates on the distal side of a constriction in its lumen—which he was the first to observe, experimentally in the dog and clinically in the human being—as in the predisposition to subclavian aneurysms when this artery rests on a cervical rib. For an understanding of this phenomenon he had sought help, he told me, from members of the physics department of the University who concluded that too many variables were involved and that if the blood vessels were replaced by rigid tubes the forces concerned could be determined.

The subject of arteriovenous fistulas had a great fascination for Dr. Halsted. Besides dilatation of the arterial trunk proximal to the fistula, he noted in patients not only that the artery was dilated proximally, but that the heart finally became enlarged, a fact which he was the first to observe. He investigated the results of congenital coarctation of the aorta with reference particularly to the dilatation which he found to occur frequently beyond the site of the coarctation.

He attempted to answer the question, "May gangrene be prevented by ligation of the vein corresponding to the occluded artery?" It was noted that gangrene occurred much less frequently in the involved limb than previously and, furthermore, that in excision of arteriovenous aneurysms of the axillary artery, there was prompt hyperemia of the forearm and hand. He asked, regarding this observation, "May not the persistence of the hyperemia be ascribed to the arterial sympathectomy?" He concluded that some degree of equilibrium of the arterial and venous systems must be maintained and that ligation of the vein raises the blood pressure in the ischemic area. He warned however, against a too general application of this principle. In conclusion it can be said, in truth, that the fundamentals established by Dr. Halsted afforded the real foundation upon which our golden era of vascular surgery has been built.

# THE IMPACT OF HALSTED UPON SURGICAL TECHNIQUE OF TODAY WITH PARTICULAR REFERENCE TO HIS OPERATION FOR HERNIA

WILLIAM F. RIENHOFF, JR., M.D., BALTIMORE, MD.

IT WAS my privilege and good fortune to have been associated with Dr. Halsted as one of his students in the medical school, as a house officer on the surgical service, and, all too infrequently, in the surgical Hunterian laboratory. Although these contacts were for a brief period only, the impact of his teaching was indelibly impressed upon me, and the truth and soundness of his conclusions have become ever clearer with the passage of time. His remarkable influence has become ever greater since his death in 1922. At the operating table, in the laboratory, or in whatever place surgery is discussed, the opinion of Dr. Halsted is forever within one's thoughts as an invisible force urging one on to improve and inquire. In a degree rarely matched, Dr. Halsted combined the interests of the practical surgeon with those of the experimental biologist in his fundamental studies of the processes of wound healing. He was one of those giants, like Hunter, Claude Bernard, and Lister, whose stature seems to grow as their figures recede into the past.

The influence of Halsted was far greater than that of teacher on pupil, master on disciple. His staff was pervaded by a cementing spirit of loyalty to him and to his very real, if indefinable, personality.

With all his important contributions to the craftsmanship and technique of practical surgery, it was the genuinely scientific attitude of mind and mode of approach to the solution of surgical problems that characterized Dr. Halsted's thinking.

The recognition and application of the principles of antiseptic surgery introduced by Lister in England were enthusiastically transferred to the practice of surgery in America, mainly by Halsted. It is safe to say that no one did more by precept and example to secure the triumph of aseptic surgery which came in later years.

Although there is marked contrast between the vigorous years of his remarkably active and successful career in New York and the later years in Baltimore, the germs of his subsequent scientific and surgical interests were planted in New York. It was in New York that he developed a system of extramural teaching called a private quiz. Much emphasis was placed on anatomic dissections in those quizzes and it was the great challenge of the cure of inguinal hernia that inspired Halsted while in New York to make elaborate studies of the fascia of the groin. The impact of Halsted upon the surgical technique of today derived more from the development of his operation for the cure of hernia than from any other of his operative procedures.

Read at the Centenary Celebration of the birth of William Stewart Halsted, Baltimore, Md., Feb. 6-9, 1952.

With the beginning of his surgical service in the Johns Hopkins Hospital Dr. Halsted's major interests came to the fore as one may easily discover from the papers presented at the meetings of the Johns Hopkins Medical Society. From 1889 to 1895 the subject of the cure of hernia came up eight times.

The evolution of the operation for the radical cure of hernia necessitated the development of the most meticulous surgical technique. This led Halsted to a careful microscopic study of wounds and the realization that care in operating, the exact approximation of surfaces, absolute hemostasis, and the avoidance of dead spaces was as important for results as the supposed avoidance of bacteria. He made a careful study of the organization of blood clot which, he showed, played an important role in wound healing, and of the causes of stitch abscess. He devised the subcutaneous suture to avoid passing the suture through the skin which could not be disinfected. It was from such studies that he learned how to treat the tissues as he made the wound that was later to heal. He treated them tenderly and watchfully, gently separating them and painstakingly transfixing with a needle and tying with extremely fine black silk every minute bleeding vessel—so that in the course of his dissection everything was left clean and dry and above all not bruised or strangled. Equally careful was his attention to the bringing together of the tissues when the wound was to be closed: fascia exactly united to fascia, muscle to muscle, layer to layer, and finally the skin with carefully placed sutures, often of silver wire, and then a superficial epithelial suture—near the surface and bringing together only the very edges, so that after a time it could be rubbed off. All this extreme care he probably exaggerated a little so that the assistants might at least approach the ideal when it came their time to operate.

Leriche says, in admiration of Halsted's precise technique, "One might think that he who conceived this must be a complex being, but one would be mistaken. Halsted is the most unpretentious man—the most exquisitely unaffected that one could find. He accomplished the rite which he has established as something commonplace—and never speaks of it. It is almost as though he thought everyone had always done just that, so normal it seemed to him."

It was thus the perfection of the operation for the cure of hernia that augmented Halsted's great interest in wound healing, and the application of his original and fundamental observations that made possible his outstanding success at a time when the most distinguished older surgeons of that era had abandoned the idea as well as the expression of "cure" in the treatment of hernia. The surgical treatment of hernia, then, must have been a real challenge and a subject worthy of the best efforts of any young surgeon, not only because of the failure of previous efforts but because of the frequency of the condition, together with its incapacitating effect, and the danger to the life of the individual suffering from it. The story of Dr. Halsted's work on the operative cure of inguinal hernia is a long one and characteristic of his tenacity of purpose until his goal was reached.

At a meeting of the Johns Hopkins Medical Society in November, 1889, Dr. Halsted presented five patients upon whom he had successfully operated

for inguinal hernia by a new method devised apparently on the basis of all those dissections of fasciae of the inguinal region which he had made in New York. This operative procedure was founded upon certain fundamental principles which have remained sound and unshaken up to the present time. These fundamental principles consisted of (1) an incision through all the tissues from the skin to the peritoneum, exposing the inguinal canal throughout its entire length, (2) the careful isolation of the vas deferens and its vessels and their later transplantation to the upper outer angle of the wound, (3) careful dissection, high ligation, and excision of the hernial sac, (4) the secure closure and repair of the anatomically weak area of the abdominal wall in the inguinal region (accomplished by the use of strong silk sutures, passed so as to include everything between the skin and the peritoneum, together with the shelving portion of Poupart's ligament), and (5) the avoidance of infection as a postoperative complication.

In the closure and repair of the anatomically weak area of the abdominal wall, the muscles and fascia were most carefully approximated to Poupart's ligament without the slightest tension. In his operation, as first described, the vas deferens and vessels of the spermatic cord were brought through the abdominal wall high up above the internal inguinal ring and well away from its natural point of emergence, the transplanted cord thus lying on top of the aponeurosis of the external oblique muscle and beneath the skin. The skin and subcutaneous tissues were closed with subcutaneous sutures to prevent infection from the skin.

Eight months after Halsted's original description of his new operation for the radical cure of inguinal hernia, Bassini, of Padua, Italy, published an account of his operation for the cure of inguinal hernia, and at that time reported 251 cases.

Dr. Halsted in his paper in *Bulletin of the Johns Hopkins Hospital,* 1893, stated, "Bassini's operation and mine are so nearly identical that I might quote his results in support of my operation."

It is interesting how often in the history of medicine, men great distances apart quite independently think of the same thing at the same time. Obviously Dr. Halsted had no more knowledge of Bassini's studies than the latter had of Dr. Halsted's, and the question of priority is not worthy of discussion.

At any rate, the number of patients requiring relief from hernia increased steadily, and in 1892, 1893, 1894, and 1895, Dr. Halsted published other papers on the results of his operation and upon the various modifications in the procedure which he had devised. In 1899, Dr. Bloodgood reviewed these results in several hundred cases, and in 1903, Dr. Halsted published one more paper with illustrations describing the operation in its best form. This procedure is performed today in almost every case. Finally, Dr. Adrian S. Taylor* reviewed, in 1920, the results of about 2,500 operations in a paper in which the value of each new detail in the operation is estimated. Through these years there had been much discussion as to the propriety of transplanting the cord, of excising the veins, of the use of the fascia of the rectus muscle, and the

*Taylor, Adrian S.: Arch. Surg. 1: 382, 1920.

especial value of the cremaster muscle in completing the closure of the weak place in the lower abdominal wall, and of the importance of high ligation of the neck of the sac. Taylor writes:

> Thus there may remain, as Dr. Halsted believes, little of the operations of Bassini and himself that is essential except the free and clear exposure of the entire canal which makes possible the high ligation of the sac. The transplantation of the cord and the futile attempt to re-establish the obliquity of the canal may be omitted. Even the suture of the internal oblique muscle to Poupart's ligament may prove to be an unnecessary detail provided the cremaster muscle is well developed and properly utilized.

The result of these modifications was really the establishment at the Johns Hopkins Hospital of two different operations for hernia: that which has been described which included the transplantation of the spermatic cord, and a second which did not include the transplantation of the cord. The latter, very briefly described, consisted in the high ligation and excision of the hernial sac and the repair of the weak area in the abdominal wall by a series of steps which overlapped the structures of the region from within outward and which include in proper order the bringing of the cremaster muscle under the lower edge of the internal oblique muscle, the approximation of the internal oblique muscle and conjoined tendon to Poupart's ligament, and the overlapping of the fascia of the external oblique muscle. The spermatic cord was left at its natural point of emergence at the lower angle of the wound. The skin was closed as in the first operation. With increasing experience it was found that of the two varieties of inguinal hernia, the indirect or more common inguinal hernia could usually be cured without the transplantation of the cord, while the direct inguinal hernia, which was much more difficult to cure, required, not infrequently, the operation with transplantation of the cord. Thus the practice became established at the Johns Hopkins Hospital of employing Dr. Halsted's first operation with transplantation of the cord for the direct inguinal hernia, and his second operation without transplantation of the cord for the indirect inguinal hernia. The final addition to the hernia operation which Dr. Halsted developed was the use of a flap of the anterior sheath of the rectus muscle, which was reflected downward and outward over the weak area of the abdominal wall in direct hernias and sutured to Poupart's ligament. This he thought would still further strengthen the abdominal wall particularly in the direct hernia which recurred more frequently than did the indirect hernia.

This, in short outline, is the story of Dr. Halsted's contribution to the treatment of hernia. In telling it there has been no intention to disregard or underestimate the contributions of others to this important subject. The point which is important to remember is that the ideas and work which led to his contributions were his own, conceived and carried out without knowledge of the work of others. They furnish another example of his approach to a problem, his persistence in arriving at a satisfactory solution, and his honesty in estimating the value of his own work. As stated by Heuer:

The studies of Bloodgood at the Johns Hopkins Hospital showed that between the years 1889-1918, 2,486 individuals were operated upon for hernia with a total mortality of seven-tenths of one per cent. Of 2,230 patients with indirect hernia, 94.4 per cent were cured, and 5.6 per cent suffered a recurrence. Of 256 patients with direct hernia, 82 per cent were cured and 18 per cent had a recurrence. These figures are striking in comparison with the results in the treatment of hernia at the time Dr. Halsted became interested in the subject. They have been bettered by some of his pupils who, using the same methods but profiting from his experience have more advantageously selected cases for given procedures, and have by better techniques lowered the total incidence of infection. The importance of postoperative infection is shown in the Johns Hopkins Hospital cases. In all operations for hernia which healed by first intention or primary union, there were only 4.1 per cent recurrences; in operations followed by suppuration there were 30 per cent recurrences.

It is, of course, a platitude to say that a good surgeon is not merely one who operates well. The qualities required to make an ideal surgeon such as Dr. Halsted are many: gifts of character, leadership, wisdom, judgment, compassion, and the finest technical skill. In respect to the latter, Halsted early realized that surgery was not only a science but an art, work demanding the highest craftsmanship and a knowledge of all the technical skills. In all his movements there was neither haste nor waste. It mattered less how quickly an operation was done than how accurately it was done. Speed resulted from his method and practiced facility, and was not his first and formal intention. It was an accomplishment, not an aim. And every movement told, every action achieved something. A manipulation, if it had to be carried out, was not half done or hesitatingly done. It was deliberate, firm, intentional, and final. Infinite gentleness, scrupulous care, light handling, and purposeful, effective, quiet movements which were no more than a caress characterized Halsted's operating as the work of an artist, and not merely of a hewer of flesh. It seemed to be his opinion that every operation, even those procedures which are now quite commonplace, should be executed not in the spirit of an artisan who has a job to get through, but in the spirit of an artist who has something to interpret or create. An operation should not only bring relief or health to the patient, but should give a glow of keen delight to the artist himself, a thrill of joy, and a sense of complete satisfaction. This, in my opinion, was Halsted's attitude.

There was no department of his work in which his capabilities were not outstanding; in more than one of them he was, by universal consent, the best. To talk about "the best surgeon in the world" is to drive discussion into the bogs of futile argumentation. There it sticks and no good ever comes of it. There is no best surgeon in the world because, of course, there can never be agreement upon how to distribute credit among the qualities. But since Halsted had, in some sense, every quality, he as nearly approaches being the complete, the perfect surgeon as many of us will ever see. He took surgery in his supple hands and lifted it bodily, leaving it firmly established at a new high level.

# DR. HALSTED AS AN ANESTHETIST KNEW HIM

MARGARET BOISE, R.N., GREENPORT, N. Y.

MY PART in today's program was assigned me before it was certain that Dr. Heuer's book would be available to you. I shall, therefore, not read from it, as originally intended, but have, instead, cast about in my memory for encounters with the Professor which might interest you.

It was in 1914 when Mrs. Harry Thomas prevailed upon Dr. Halsted to grant me an interview at his home on a Sunday morning. I did not realize the extraordinary privilege conferred upon me for I knew about the Professor only that he was surgeon-in-chief of the Johns Hopkins Hospital. Mrs. Thomas and I hoped that my three years' experience in anesthesia in New York might secure me a position at Johns Hopkins.

I was directed up to the second floor of the large brick house on Eutaw Place and there I entered the library which so impressed me at a glance that I still recall the atmosphere of warmth, the beautiful mahogany and colorful rugs. From a comfortable chair in front of an open fire the Professor rose to greet me and after asking me to be seated he resumed his place by the fire. He did not make it easy for me to state my case but listened attentively. His eyes, meanwhile, seemed to penetrate my mask of assurance to the quaking me beneath it.

At the end of the recital of my accomplishments in anesthesia he rose and, as he led me to the door, said, "My interest is in someone who will advance our knowldge of anesthesia. Thank you. Good morning."

In spite of his apparent lack of interest in my experience in anesthesia, Dr. Halsted consented to a plan proposed by Dr. Crowe and Dr. Young to employ me as their special anesthetist. After giving their anesthetics for almost two years, I had my second interview with Dr. Halsted, this time at his request. He offered me the position of anesthetist for general surgery. Mrs. Thomas was, perhaps, more elated over this turn of events than I, for I had enjoyed the friendly relationship with Dr. Crowe and Dr. Young who released me with the proviso that that they could call upon my services when necessary. There are some who say that Dr. Halsted had been favorably impressed by my anesthetics and was glad to reverse his opinion regarding my suitability for the job. I have always inclined toward the belief that my appointment was due to the fact that the surgical staff was being depleted by the war and that residents and interns could not be spared for anesthesia.

I was never quite sure that my work satisfied Dr. Halsted for I do not recall that he ever voiced approval or disapproval. On painful occasions when I was unable to relax a patient whose peritoneum he was trying to close, he would stand and wait and if I ventured a look over the screen I would

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find his eyes fixed upon me without the usual kindly expression and his attitude depicting exasperation. Once, at such a time, he backed away from the table and retired to his office, leaving his assistant to complete the closure. I felt in disgrace.

In the course of time Dr. Halsted allowed me to train graduate nurses in anesthesia and thus started the school which has supplied anesthetists to many hospitals. He also encouraged interns to give anesthetics under my supervision and asked me to teach the medical students during their course in surgery in the Hunterian Laboratory. There I had the opportunity of observing the Professor's love for dogs and his gentle treatment of them. One day I took my sick fox terrier to the laboratory to have Mike, the diener, look him over, but it was Dr. Halsted who took the dog out of my arms and examined him with his gentle, deft fingers. When he reported to me that he had found an inoperable tumor, he seemed as concerned and sympathetic as though it had been a member of my family, and he asked about the little dog frequently thereafter.

My veneration and affection for the Professor grew with the passing years and I have often regretted that I had not been the anesthetist during his more active years at Johns Hopkins, when he was making his great contributions to surgery, or in the early days in New York so graphically portrayed by Dr. Heuer. One passage in Dr. Heuer's book gives an interesting commentary on surgical practice in New York City in 1880. Dr. Halsted at that time was entrusted by Dr. Sands with the purchase of instruments for the New York Hospital. In one order to Europe he listed twelve artery forceps.

"What shall we do with so many?" was Dr. Sands' amused question.

No hospital in New York then had more than six artery forceps at a time. Two years later Dr. Halsted began practicing his operation for cancer of the breast. He must soon have justified his extravagant order for twelve forceps.

In 1921, a dinner was given in honor of Dr. Heuer and Dr. Reid who were leaving for Cincinnati. Dr. Hal Thomas called for me and stopped at 1201 Eutaw Place for the Professor. To my embarrassment, Hal told the Professor that I had an idea—a word which always seemed to intrigue Dr. Halsted. He listened attentively to my belief that fear during the induction of anesthesia liberated a substance in the blood of the patient which was antagonistic to ether and gas, and that I thought it might be adrenalin. The Professor encouraged me to continue my observations but stated that there was no way of detecting an increase in adrenalin in the blood in so short a space of time.

"You may be right and the future may prove you so," he said.

The sympathetic portrayal of his chief was not a difficult task for Dr. Heuer. The book might have been published years ago had it not been for the chapter on cocaine. In his desire to make an irrefutable statement to the effect that Dr. Halsted had been totally and permanently cured of his addiction every possible clue was followed up. Word would come from one source that no one could doubt that the Professor continued to take cocaine throughout his lifetime; another would be equally positive that he was cured

of his addiction early in the Baltimore days. A conclusive answer to the question was not found and, according to Dr. Heuer, it never will be found. The chapter was written and rewritten many times and was always a stumbling block. In his last year Mrs. Heuer and I tried to convince him that he need only relate all known facts and leave it to the individual reader to judge for himself which road the Professor pursued: The courageous and victorious fight against the enemy, or the equally courageous thirty years of fruitful activity with the haunting enemy always at hand.

In reading George Heuer's book it will be apparent to you that he did not deviate far from the road he thought Dr. Halsted would approve. I am not able to judge how far Dr. Heuer traveled along that road but, like many gathered here today, he went faithfully forward on the way projected by his illustrious teacher, Dr. Halsted.

# THE STORY OF HARVEY CUSHING'S APPENDIX

SAMUEL CLARK HARVEY, M.D., NEW HAVEN, CONN.

IN THE autumn of 1934, the Society of Clinical Surgery met in New Haven, due, in part at least, to Harvey Cushing having, in 1932, returned to Yale where he was to enjoy some seven years of freedom from administrative and clinical responsibilities. They were indeed happy years, marred only by a progressive arteriosclerosis, first manifesting itself in his feet, then in a gastric ulcer, and finally in a fatal coronary occlusion. These vicissitudes he bore with extraordinary patience and fortitude, especially notable when one keeps in mind that he was a physician who, in the pursuit of his professional activities, had given little evidence of suffering gladly fools or other impedimenta.

It was upon his admission to the hospital in February of 1934 that I learned that he had had an appendectomy some thirty-seven years previously.[1] Over the right lower rectus was a lengthy, well-healed, longitudinal scar with a weakness of the median half, which was the first evidence, but much more spectacular was the appearance in the roentgen films, taken because of his gastric ulcer, of a gridiron of several sutures of silver wire placed transversely, all of which were broken.

In the past history taken at this time it was noted that "in 1897 at the Johns Hopkins Hospital he was operated upon by Drs. Halsted and Finney for acute appendicitis and was back at work within two weeks."

This aroused no particular curiosity until, on sending out the notices for the meeting of the Clinical Surgical Society, Dr. Bloodgood replied by regretting that he could not attend the meeting, going on to say,

> I am delighted to hear again from you that Cushing is coming on splendidly. I should judge that this is the first time that he has really ever given his health any consideration. When we operated upon him for appendicitis more than 35 years ago he insisted upon lying on his stomach and the wound broke down, but fortunately only superficially. When Dr. Halsted urged immediate operation for appendicitis Dr. Osler advised delay and we had the greatest trouble in getting Dr. Osler and Dr. Cushing to consent to this operation. These are delightful idiosyncrasies but sometimes have a dangerous element. Nevertheless these characteristics may have been behind the tremendous push that has made his life as a surgeon such a great benefit to humanity, and I hope that the rest of his life in Yale University will be free from the annoyances that must come to the head of every department. I hope you will succeed in keeping him there in good health for some years.[2a]

I sent this letter to Dr. Cushing with the thought that he would be pleased at Dr. Bloodgood's kind and continuing interest in him, but also, I am afraid, somewhat mischieviously with the hope that there might be some further amplification. In this I was not disappointed as the following letter shows.

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Read at the Centenary Celebration of the birth of William Stewart Halsted, Baltimore, Md., Feb. 6-9, 1952.

> Thanks for letting me see Joseph's letter. It's a perfect libel about my appendix operation. It shows how people's memories of things are what they want to remember. They had operated on only about half a dozen cases of appendicitis at the J.H.H. in 1896 and never until an abscess had formed. I knew perfectly well what I was in for, having seen a good many operations in Boston. If it hadn't been that I had been taught to count leucocytes and persuaded W. G. MacCallum, then a fourth year medical student, to count mine during the evening and morning in the course of which they went up to 34,000, they wouldn't have operated upon me at all. All the people were going to a champion baseball game that afternoon and they were very much annoyed to stay home. Halsted, Finney and Bloodgood did the operation and they had all six hands inside of me at once—big hands at that. Hugh Young gave me chloroform. I was all for the operation from the evening before, but they didn't get round to it until about noon the next day.
>
> They used a mid-rectus incision from ensiform to pubis and got mixed up in the epigastric artery which they had to tie twenty times with heavy black silk ligatures. All these ligatures came out in the course of the next two or three months.
>
> It's just like Joseph to ascribe my broken-down wound to lying on my stomach which I couldn't have done if I had wanted to for the next two months. No self-respecting wound would have held after the way they had mussed it up.
>
> I want to get this story straight lest Joseph develop this idea of my delightful idiosyncrasies.[2b]

It seemed apparent that this operation was not as routine as had at first appeared and my curiosity was aroused as to what really had gone on. Therefore, through the courtesy of the record room of the Johns Hopkins Hospital, I obtained a typescript of Dr. Cushing's case history[3] and recently, due to the kindness of his biographer, Dr. John Fulton, a photostatic copy has been at hand.

These are at the same time enlightening and confusing for, aside from two sentences in parentheses in the past and present history and a final summary report of certain personal observations of the patient, the manuscripts are not signed. However, the manuscript in which these appear is very definitely in the hand of Cushing and gives a play-by-play description of what went on up to the time of the operation. It seems apparent that this was written during his convalescence when his innate urge for writing had returned to its normal level for, in the script, reference is made to what had been found at the operation. He also wrote a letter on the fourth postoperative day to his father, and during this period he composed his only known attempt at prosody, the "Efur" poem.[4]

In addition to the portion of the history undoubtedly written by Cushing, a brief operative note containing a description of the pathology findings is a part of the record, but not in his hand. Following this are several brief postoperative notes, the first made on the day of the operation which says, "There was more pain referred to the wound than usual after operation." On the fifth postoperative day is recorded in some greater detail the evacuation of a superficial hematoma. It seems probable that these, as well as the operative note, were written by Halsted. On an additional sheet there are also notations made by Cushing on the fifth, seventh, twelfth, and nineteenth postoperative days, the last being that of discharge from the hospital. Finally there is also a pathology note with a detailed description of the appendix and a rather crude

sketch of it opened and containing an "enterolith." This is in a hand differing from those preceding and is probably by Bloodgood. There is a short final summary by still another hand saying that the sinus was closed in three months.

The final "follow-up note" made at autopsy 42 years later by Dr. Harry Zimmerman of Yale is: "A narrow but tough fibrous adhesion was seen between the cecum and the anterior abdominal scar, the residuum of an appendectomy. The greater omentum covers the intestines in apron-like fashion without being adherent to any viscus."[1]

Several aspects of these records make them of particular interest. The first of importance, perhaps, is the patient's own account of the incident at the time it occurred, and again after thirty-seven years in his letter, the former of course being the more authentic. The latter seems to show a certain degree of rancor at the course of events, which may have been slumbering subconsciously but probably not to the degree expressed, for there was often a considerable poetic license when Dr. Cushing presented a story. It is true, for instance, that Halsted, Finney, and Bloodgood were directly involved in the operative procedure and also that the hands of each were not of the type popularly and incorrectly supposed to be characteristic of a facile surgeon. For that matter, those of Cushing were not either, his bearing the residual effects of his baseball days. Perhaps the fact that he had the captains of a famous Yale football team and of an equally distinguished Princeton team presiding over his destiny at this critical time should have softened this criticism, but in any case it is not of major significance. The fault, if there be such, lay elsewhere.

An astute analysis of the signs and symptoms led Halsted to predict exactly what he found, a free and diseased appendix lying deep in the pelvis. For this he needed ample exposure and in getting this by a split rectus incision he, as is so often the case in this approach, got into trouble with the deep epigastric vessels. He states himself that the "deep epigastric was ligated." The notation as to the closure does not mention silver wire; it reads, "Six mattress sutures in muscles, 1-2 cm. apart." Were it not for the roentgen film taken in 1934 and Cushing's notation on the fifth postoperative day, "Silver suture removed without causing particular pain," one would not know that the deeper layers were closed with burried silver wire. Putting all the facts together it would seem possible that one or more of these broke immediately after the operation, leading to the hematoma and the pain in the wound. This would likewise account for the persisting sinus and the discharge of silk sutures. That these were so many and so large as suggested in Cushing's letter may be questioned; they always seem that way to the one most intimately concerned, that is to say, the patient.

The course of events preceding the operative procedure, as stated by Cushing clearly and concisely, is accompanied by a sketch indicating the "point of tenderness" quite below and to the inside of McBurney's point. The "onset" was at four in the afternoon "after a wretched dinner," with progressive abdominal cramps, which by 9 P.M. were "pretty constant and located below the umbilicus" although the "localization" was never definite and never marked in the right iliac fossa. By 5 A.M. tenderness was quite distinct on relief of pressure and a similar discomfort was felt on evacuation of the bladder.

There is no indication in this note of anyone having been aware of his illness up to this time, although shortly after the onset he "took 1/6th gr. of calomel," and at 5 A.M. 1/10 gr. of morphine. It seems improbable that he was seen by Halsted or Osler on a Sunday evening, or at any time previous to their appearance at the accustomed hour the following morning. On the other hand, although Cushing had shortly before this episode been appointed resident, Bloodgood was remaining on for another year to finish up his review of herniotomies at the Johns Hopkins Hospital that appeared in the *Bulletin* of 1899. He continued to live in the hospital and it is possible that he was aware of Cushing's condition and recommended immediate operation while Cushing, agreeing in principle, chose to stall until Halsted and Osler arrived on the scene. In any case, it is noted that at 8 A.M. the following morning Strong did a leukocyte count and found 13,000. The temperature was taken then and registered at 101° F. There was "absolutely no muscle spasm although the patient found it impossible to straighten up when on his feet." It is probable that it was at this time that he was officially admitted to the hospital as a patient. One hour later, 9 A.M., Halsted and Osler were in consultation and did not advise operation. They undoubtedly delayed for further observation and a check on the "white count" which was done at 12 noon, this time by W. G. MacCallum and found to be 23,000. The temperature then was 101.6° F. and Dr. Halsted gave the patient 1/6 gr. of morphine presumably preparatory to the operation.

Cushing summarized the reason for the decision as follows:

> This steady *elevation* of *temperature,* c.f. Pulse not affected, without remission and increasing of leucocytes were practically the only definite guides to the local condition found. C. *Vomiting.* Pain though severe was never either in site or severity characteristic. *Tenderness,* never was marked or at the usual site. *Muscle spasm* was practically absent till a very short time before the operation.

From this record, written by Cushing himself, it appears that the course of events did not differ materially from what might happen today. The calomel, 1/8 gr., and morphine, 1/10 gr., may have served to obscure the picture somewhat, but the small doses employed were probably not very effective. The delay in bringing in the senior surgeon and physician, even in well-organized services, might easily occur, for the resident is somewhat loath to impose his own afflictions upon his superiors, especially in the middle of the night. Moreover, it is significant that when Halsted and Osler did arrive, they were not sufficiently certain to proceed at once with an operation, but chose a period of observation.

One may infer—as both Bloodgood and Cushing did in retrospect—that positive action should have been taken much earlier, and that this was due to a lack of knowledge of, and experience with, appendicitis on the part of those concerned. However, one should consider first these attributes in respect to the prime performers in the incident:

Cushing, as a student from 1891 to 1895 at the Harvard Medical School,[5] had as his teacher in Pathology Fitz, whose classical paper, "Perforating Inflammation of the Vermiform Appendix; with Special Reference to its Early Diagnosis and Treatment," had appeared in 1886[6] and a second paper, "Appendicitis,

some of the Results of the Analysis of 72 Cases seen in the Past Four years,'' in 1890.[7] In 1888, John W. Elliot of Boston had published an account of an operation upon Dr. Alfred Worcester of Waltham, Massachusetts,[8] who, his biographer Ernest A. Codman states, ''was not only the first person in this community (Boston) to have his appendix sought by the transperitoneal route but was one of the first men to have his abdominal cavity opened for any operation.'' An abscess was found and drained but the appendix was not uncovered. Dr. E. R. Cutler who assisted in this operation seven weeks later ''did the first successful removal of an appendix prior to its perforation.''[9]

Following his graduation, Cushing served a year in the Massachusetts General Hospital where the attending surgeons were avidly developing the field of appendicitis. By this time, here and elsewhere, the fundamental principles involved were fairly well established. It is not surprising, then, that Cushing may have brought to the Johns Hopkins Hospital an understanding and experience that made him somewhat critical.

As a background of the picture in Baltimore in 1896, it is well to keep in mind that the hospital had been open for only seven years and that the medical school was to graduate its first class in 1897. The attending and teaching staffs were new in Baltimore, and it required time to establish a reputation that attracted patients, to obtain and develop personnel, and to perfect an organization.[10] This was accomplished with remarkable facility, but there was not, in 1897, tradition and maturity such as obtained at the Harvard Medical School and the Massachusetts General Hospital. Until the school began to graduate students, there was no autogenous source of professional personnel.

So it was that those concerned in Cushing's episode, except for the two students who were to graduate in the first class, MacCallum and Strong, had obtained their medical education elsewhere—Osler[11] at McGill, Halsted[12] at the College of Physicians and Surgeons in New York, Finney[13] and Cushing[4] at Harvard, Bloodgood[14] at the University of Pennsylvania, and Young[15] at the University of Virginia.

TABLE I

| | | YEAR OF GRADUATION | AGE IN 1897 |
|---|---|---|---|
| Osler | McGill | 1872 | 48 |
| Halsted | Physicians and Surgeons | 1877 | 45 |
| Finney | Harvard | 1888 | 34 |
| Bloodgood | University Pennsylvania | 1891 | 30 |
| Cushing | Harvard | 1894 | 28 |
| Young | University Virginia | 1894 | 27 |
| Strong | Johns Hopkins | 1897 | 25 |
| MacCallum | Johns Hopkins | 1897 | 23 |

So far as the background of these physicians in respect to appendicitis goes, there will be considered first their experience previous to the opening of the Johns Hopkins Hospital and then that in this institution up to 1897, the time of Cushing's operation.

Osler, the senior member of the group, while at Montreal and Philadelphia as a pathologist described instances of ''perforated appendicitis'' observed at

autopsy.[16] Following the appearance of Fitz's paper he wrote a lengthy but concise editorial in *The Medical News* of Philadelphia, which gives the gist of the article and in it makes this comment:

> The treatment of this affection has been too much in the hands of physicians, and in too many cases there is an unjustifiable delay while waiting for the more definite signs of suppuration. If good is to be obtained by an operation, the earlier it is done the better chance has the patient for recovery. Fitz very correctly states that if, after the first twenty-four hours from the onset of the severe pain, the peritonitis is evidently spreading and the condition of the patient is grave, the question should be entertained of an immediate operation for exposing the appendix and determining its condition with reference to its removal.[17]

I can find no evidence other than this of any particular interest in clinical appendicitis until he transferred to Baltimore.

There Osler shortly turned to composing his textbook, *The Principles and Practice of Medicine*, the first edition appearing in 1892.[18] In this, a section is captioned, "Appendicitis" with "Typhlitis and Paratyphlitis" in parentheses. Some seven pages are devoted to it, of which about one-half have to do with the anatomy and pathology. The symptoms, diagnosis, and prognosis are centered around "catarrhal" and "perforative" appendicitis; they take up the remaining space, save one page for treatment. In the last he discusses medical treatment, which is warned against, and limits the function of the physician to saying "whether the case is suitable and when the operation should be performed." The indications for this are not clear and he recommends no operation in "recurrent appendicitis," unless the physician can "hand over his patient without qualms to a modern operator whose success has demonstrated the safety of his methods."

In the second edition, that of 1895,[19] this chapter is, as Osler himself states, "completely rewritten," with twice as much space being used. The section dealing with anatomy and pathology has been somewhat reduced, while the remainder is devoted to the clinical disease. He gives credit to American physicians and surgeons for bringing about the recognition of the importance of appendicitis, naming Pepper and Fitz as the primary instigators and then a number of surgeons: Willard Parker, Sands, Bull, McBurney, and Weir of New York—all earlier colleagues of Halsted—Morton, Keen, J. William White, and Deaver of Philadelphia, Senn of Chicago and, lastly, Treves of London. He also refers to five monographs which he had consulted. The authors of these were: Fowler (1894), American, Kelynack (1893), and Hawkins (1894), English, Telamon (1892, translated into English in 1893), French, and Sonnenberg (1894), German. Boston surgeons are completely overlooked, including J. W. Elliot[20] and A. Worcester[21] and the papers they wrote in 1892. Perhaps this blind spot was corrected after Cushing arrived, for he had worked with Elliot at the Massachusetts General Hospital as well as with A. T. Cabot, Cutler, Richardson, and Porter who had also made important contributions to the knowledge of appendicitis.

Osler's change in position is shown in the opening sentence of the section on "Treatment" which reads:

> So impressed am I by the fact that we physicians lose lives by temporizing with certain cases of appendicitis, that I prefer in hospital work to have the suspected cases admitted directly to the surgical side.

He states, also, "There is no medicinal treatment of appendicitis," and, "The mortality from early operations under these circumstances is very slight." Unfortunately, "these circumstances" were, and this is in italics, "*when by the third* (sic) *day the features of the case point to a progressive lesion.*"

The third edition, that of 1898,[22] has minor changes only in the section on appendicitis, and is therefore essentially the same as the second.

While Osler transferred by choice from Montreal to Philadelphia and then to Baltimore, each step offered him additional opportunity. The story is somewhat different with Halsted. The years in New York from 1877 to 1885 were, after two of study in Europe, those of furious activity and concentration of effort directed toward a career such as his associates, George E. Brewer, William T. Bull, Ellsworth Elliot, Frank Hartley, Charles McBurney, and Francis H. Markoe later achieved. This was abruptly ended in 1885 and after a troubled interlude he went to Baltimore in December of 1886. From this time until the hospital was opened in 1889, he worked in Dr. Welch's laboratory, quite set aside from clinical responsibilities. This mischance and the years in the laboratory changed him philosophically and professionally into the surgeon for whose qualities we honor him today.[12]

In the period from 1885 to 1889 one would not expect him to have had much experience with appendicitis and, so far as I can determine, there is no evidence of such experience. His previous clinical activities fell in the time of Weir's early contributions and before the clinical paper of Fitz.[6] The only reference to appendicitis is given in his own words, and reflects accurately the situation at that time.

> Dr. Hackley, recalling later my eagerness to operate early in cases of perityphlitis, assigned to me in the early eighties two private patients with acute general peritonitis. Assisted by Richard Hall, I removed the appendix promptly in each case, but with fatal result. These were the first appendectomies performed in New York. Fitz's paper appeared a few years later (1886). Hall reported subsequently a successful appendectomy, the first in the country. He was operating for the relief of an "inflamed hernia" and found the inflamed appendix in the sac. The main surgical topic with Sands, Hall and myself in the early eighties was perityphlitis. We (Hall and I), unable to secure a patient, urged Sands constantly to operate and remove the appendix as soon as possible after the appearance of symptoms. The early cases were only seen in private practice of which I had none. McBurney in his first paper (1889) stated that he assisted Sands in performing six appendectomies prior to his own operations. If Sands had lived he would undoubtedly have anticipated McBurney but this does not detract from the great credit due McBurney for early diagnosis and operation in the interval.[23]

At the Johns Hopkins Hospital only two papers by Halsted appeared bearing directly on appendicitis, though there are several minor references in others, of no particular moment for this discussion. The first of the major contributions was presented at the Johns Hopkins Hospital Medical Society in December of 1893 and is entitled "A Diagnostic Sign in Appendicitis."[24]

This sign he believed to be "probably present in all cases and in all stages" save in the presence of general peritonitis with marked distention. It is the inability to palpate successfully the iliac fossa because of muscle spasm, or adhesions, or exudate. He notes that muscle spasms may be diminished by counterirritation with the Paquelin cautery or obliterated by deep anesthesia. He goes on to comment on the importance of "the packing off of the uninfected portion of the abdominal cavity from the infected portion prior to opening of an abscess" and describes in detail just how he goes about it. He then says:

> We have operated upon thirty-four cases of appendicitis, and without a death if we except the nine perfectly hopeless cases which had acute suppurative peritonitis before they were operated upon. Five cases of appendicitis with a less desperate form of peritonitis were saved by operation. Of four cases of appendicitis without peritonitis operated upon by me outside of the hospital, all recovered from the disease; and of the two with general suppurative peritonitis, both recovered from the peritonitis, but one of them died from hemorrhage about two weeks after the operation and when he was believed to be surely convalescing.

The second paper is entitled "A Postscript to the Report on Appendicitis."[25] This title is somewhat confusing unless one is aware that Finney on April 2, 1894, had exhibited before the Johns Hopkins Hospital Society seven cases of appendicitis cured by operation, and had at the same time discussed statistically the over-all experience there, together with individual instances illustrative of the various forms of the disease as well as the indications for operating and the operative procedure.

One of the patients presented had a hernia and Osler, who was present, asked, "What is the objection to closing the wound in these instances of appendicitis unless there is extensive suppuration?" and commented on a physician in New York who was treated by the "open method" and had a large incapacitating hernia.

Finney closed the discussion by saying that the objection was fear of infection where the abscess cavity could not be disinfected, and that he would, under suitable circumstances, undertake a closure.[26]

The presentation of April 2 by Finney was published in the *Bulletin of the Johns Hopkins Hospital* of June-July, 1894. Apparently Halsted was not present at the presentation and on reading the account of it he was stimulated to write the "Postscript" which appears in the November issue.

It opens as follows: "Dr. Finney's reports on the treatment of the wound in cases of appendicitis have been abbreviated so much as possibly to mislead those who are not familiar with our methods." He then portrays precisely how the gauze drainage is placed and the closure of the wound about it. He goes on to describe the suturing of such a wound, and in a footnote states that he presented a case of appendicitis to illustrate how this is done. In this instance he used "buried sutures of silver wire . . . to bring together the cut edges of the abdominal muscles . . ." He decries attempts at disinfection of an abscess cavity within the peritoneum and refers to experimental work of his which "demonstrates conclusively the result of innoculation of strangulated tissues in

the peritoneal cavity.'' He also describes the incision used, and the reason for it, as well as the methods employed to prevent contamination of the peritoneum.

It is disappointing that there is no further discussion of appendicitis by Halsted, but it is apparent that he had mastered the fundamental principles involved and left to others the flooding of the literature with case reports and competitive scores as regards the number of appendectomies done.

It is of interest, however, to know the quantity of material from which he derived his knowledge and judgment. There are three overlapping reports appearing as follows: one by Halsted, December, 1893,[24] one by Finney, April, 1894,[26] and one by Bloodgood, June, 1895.[27]

TABLE II

| REPORTER | NUMBER OF CASES | GENERAL PERITONITIS | ABSCESS | ACUTE AND RECURRENT APPENDICITIS | OPERATIONS | DEATHS | OPERATIVE MORTALITY (PER CENT) |
|---|---|---|---|---|---|---|---|
| Halsted (Dec. 18, 1893) | | | | | | | |
| Hospital | 34 | 9 (9)* | 5 (0) | 20 (0) | 34 | 9 | 26.5 |
| Outside | 6 | 2 (0) | | 4 (0) | 6 | 0 | 0 |
| Total | 40 | 11 | | | 40 | 9 | 22.5 |
| Finney (April 2, 1894) | | | | | | | |
| Hospital | 35 | 8 (8) | 11 (2) | 16 (0) | 35 | 10 | 28.5 |
| Bloodgood (June 1, 1895) | | | | | | | |
| Hospital | 53 | 10 (10) | 17 (1) | 24 (1) | 51 | 12 | 23.5 |

*Figures in parentheses are deaths.

It would also be of interest to know the number of cases between Bloodgood's report and Cushing's operation but, for accurate data concerning this, one would have to scan the records of these years. For the purposes of this discussion, it is perhaps sufficient to extrapolate from the figures at hand; in doing this a minimal increment for two years would be about thirty-seven instances, or a total of some ninety since the opening of the hospital. Certainly Cushing's estimate in his letter of a ''half dozen'' was somewhat of an understatement. Perhaps his experience with a patient whom he had operated upon less than a month previously, who had had a ruptured appendix with peritonitis, and who died ten days later, may have cast a shadow over the past as well as the immediate future.

The third physician in seniority involved in the episode was Finney. It is not clear that he had any part in it, beyond that of assisting at the operation. He had come to the Johns Hopkins Hospital at its opening in 1889, following some two years as a house officer at the Massachusetts General Hospital. Like Cushing he had graduated from the Harvard Medical School, but some years earlier, and his own acquaintance with the disease therefore extended backward into the time when Fitz brought it sharply into focus.

In his autobiography, Finney expresses satisfaction that in 1886 Fitz examined him, made a diagnosis of appendicitis, and said, ''If he isn't better

very soon, you had better call in Dr. Porter''—A surgeon attending at the Massachusetts General Hospital.

Fortunately, so far as he was concerned, he improved and was not operated upon. Two years later he had the opportunity to sit in on a consultation with Fitz and Homans in which the former wished to delay and the latter to operate. The advice of Homans was taken. In this case, however, the patient had a ruptured appendix and a spreading peritonitis from which he died.

Finney says of this period, ''Very few operations were being done for the disease at the time that I was in the Massachusetts General Hospital, and then in the vast majority of instances only after the appendix had ruptured and an abscess had formed. The usual operation consisted of simply draining the abscess.''[28]

When Finney came to Baltimore, Halsted turned over the Surgical Dispensary to him, and also used him as an anesthetist and a first assistant. When Halsted was absent, as was not infrequently the case, he filled in as a substitute senior surgeon and continued with these responsibilities for many years while carrying on an extramural practice. We still see him assisting Halsted in an operation being performed at the opening of the ''New Surgical Amphitheater'' in 1904.[29] It seems probable that he was responsible for some of the operations done for appendicitis in those early days and had a part in the developments then taking place. In any case, it is certain that he was well informed as to Halsted's views and also those of Osler.

Lower down in the hierarchy comes Bloodgood,[14] who, after spending a year as resident physician at the Children's Hospital in Philadelphia, became an assistant resident at the Johns Hopkins Hospital for a few months and then went abroad for a year. On returning he was appointed Resident in Surgery and served as such for four years. During his last year, Cushing was an assistant resident, and then followed him as resident. So far as Bloodgood and other members of the house staff were concerned, one can, I think, safely assume that Osler's *Principles and Practice of Medicine* was, in its own birthplace, as oracular as it rapidly became elsewhere, and that those working with Halsted absorbed an adequate understanding of the problems of appendicitis.

Having discussed the dramatis personae of major rank, one cannot overlook some of lesser importance who appeared briefly on the stage. One of these is the anesthetist, Hugh Young,[15] who came to Johns Hopkins Hospital in 1894, fresh from medical school, and was promptly assigned to the Surgical Dispensary. One of his functions was that of ''pouring ether'' or, in this particular instance, chloroform. At this time he was an assistant resident, but within the year was assigned by Halsted, without the asking, to head urology.

Two senior medical students also have their parts, Richard P. Strong,[30] and W. G. MacCallum.[31] The former was to become distinguished in tropical medicine and the latter equally so in pathology. They did the ''white counts,'' the results of which undoubtedly had something to do with the decision to operate. Both of them, as senior students, were working on the wards and also in the adjacent laboratories.

MacCallum was familiar with the work of William S. Thayer[32] who had come from the Massachusetts General Hospital in 1890, and was resident physician at the Johns Hopkins Hospital from 1891 to 1898. Particularly was this significant in relation to hematology, for he had spent the better part of a year studying in Germany, and specifically with Ehrlich who had perfected the staining of the leukocyte. Coming to work with Osler, whose interest in the corpuscular elements of the blood was long-standing and intense, he at once embarked upon a study of these elements that quickly established his reputation in the field. The section on diseases of the blood in the first edition of Osler's *Practice* was, in considerable part, his and the charts entirely so.[33] These charts portrayed qualitative determination of the leukocytes over successive days in various diseases of the blood; it is, then, apparent that blood counts were commonly done at the hospital for some time antedating the Cushing episode. Further evidence of this is the studies made there by J. S. Billings, in 1894, concerning the leukocytes in "croupous pneumonia," and in "malarial fever."[34] In connection with the latter, MacCallum was, as a student, studying the hematozoa of malaria, and published, in 1898, an outstanding contribution to the life history of these organisms.[35] It is, however, quite possible that he had not learned the technique of counting the blood cells until Cushing taught him, for as a student he may have missed the initial course in "clinical microscopy" given by Thayer in 1895-1896, presumably for the second year class.[32]

This may likewise be true of Strong, whose experience was parallel to that of MacCallum. He also, as a senior student, was working in the laboratory but in the field of bacteriology, presumably under Simon Flexner with whom he laid the foundation for his future in tropical medicine. Whether he also learned the technique from Cushing may be open to question, but his association with him was intimate, for on the second postoperative day he wrote a reassuring letter to Mrs. Cushing, introducing himself as "one of your son's assistants on the surgical staff here."[36]

In any case it would appear that Cushing was familiar with the significance of this procedure in appendicitis. There is no indication, so far as I have been able to determine, that other members of the staff were, antecedent to his arrival in Baltimore. In none of the three earlier editions of Osler's *Practice* is there any pertinent reference, and in the fourth, that of 1901, it is covered in a brief statement: "Severe cases usually show a leucocytosis of 15,000 to 24,000."[37]

Bloodgood,[38] in a paper delivered before the American Surgical Association in 1901 as a part of a symposium on the blood in relation to surgical diagnosis, gives the experience at the Johns Hopkins Hospital with leukocytosis in appendicitis. Unfortunately, there is no clear indication as to when this procedure was initiated, though the instances of all varieties of appendicitis up to 1901, as reported, were some 100. Among these, three instances of "gangrenous appendicitis" are reported in detail, the last being that of Cushing, although from internal evidence it would seem probable that this was chronologically first.

In a discussion of leukocytosis in intestinal obstruction, Bloodgood states, "Dr. Harvey Cushing I think was the first in this country to call attention to leucocytosis associated with intestinal obstruction."

This was in reference to a paper entitled "Laparotomy for Intestinal Perforation in Typhoid Fever" published in December, 1898, and Cushing's report of an instance in which, with a leukocytosis of 20,000, the difficulty on exploration was found to be intestinal obstruction. The detailed records of the four cases operated upon are characteristic of Cushing but even more notable are the numerous determinations of the leukocytes.[39] This may be contrasted with an article by Finney appearing in March of 1897 in which he reviews in detail some fifty-two reported cases of typhoid perforation of which three were his own. In only two of these were the leukocytes counted and, significantly, one of these was Finney's and the other that of C. B. Porter of Boston. The former patient was operated upon in August of 1895, Finney having seen him in consultation with Thayer. There was an initial count of 3,000 going to 16,400 after the perforation. The latter patient was operated upon in October of 1896 with a similar count: 6,500 going to 14,700.[40]

It is apparent that in leukocytosis following a perforation in typhoid fever as well as in that occurring in intestinal obstruction, the surgical staff of the Johns Hopkins Hospital was alert to the value of the quantitative determination of the leukocytes.

Both Cushing and Finney refer in their papers to the monograph, "The Clinical Examination of the Blood" by Richard Cabot of Boston which had appeared in January of 1897.[41] This was dedicated to Thayer and remained for several years the most authoritative text in English in this field. Cabot graduated from Harvard in 1892, three years before Cushing, and at once began his service as an intern at the Massachusetts General Hospital.

A year and a half later he presented a paper which opens as follows: "The material for this paper consists largely of blood counts, 332 in all, which I have made at the M.G.H. within the last year." Of these, 24 were in cases of appendicitis. He suggested that this technique might be of value in distinguishing between colic due to constipation and "true appendicitis." In three instances of terminal peritonitis there was no leukocytosis.

In conclusion he says, "The number of cases consulted among the surgical affections are too few to warrant any conclusions; but they are sufficient, it seems to me, to make it important that the subject of leucocytosis in surgical cases should be studied much more carefully, with the view to its practical diagnostic value."[42]

Ironically, this is followed in the same journal by a report of John W. Keefe of Providence entitled, "Twelve Consecutive and Successful Operations for Appendicitis" of which nine were in the stage of abscess. There is no reference to the use of the blood count in any of these.[43]

When Cabot published his monograph in 1897, not only had the first and second editions of Osler's *Practice* appeared but also Thayer's classic monograph on malarial fevers and typhoid. By this time Cabot had accumulated an experience, as stated in the preface, based upon "nearly four thousand blood examinations" of which about three thousand were made by medical interns, the rest by Cabot himself. He reports two instances of typhoid with perforation in which marked leukocytosis was present, and seventy-two of appendi-

citis. The analysis of this experience and the conclusions derived from it are well worth reading today and little, save the differential count, has been added over these intervening years.

It cannot be doubted that Cushing, as a student at the Harvard Medical School and as an intern in the Massachusetts General Hospital, was quite familiar with this development at the hospital, and it is highly probable that he had learned the technique involved—if not directly from Cabot, then from the medical interns who were employing it on the surgical wards. After he arrived at the Johns Hopkins Hospital, it would be consistent with his latter-day insistence that the surgical house staff carry out the diagnostic procedure if he himself made such counts and taught the students and interns working under him on the surgical wards to do likewise. This would seem to fit in with Strong and MacCallum making their observations in his case, and with Bloodgood's early and continuing interest in leukocytosis in surgical diseases.

The pursuit of this story arose from the letters of Bloodgood and Cushing, whose thrusts and counterthrusts need not be taken too seriously, nor, I suspect, were they intended to be.

In any case, one must conclude, I believe, that Harvey Cushing was most fortunate in having his appendicitis in an institution where this disease was so well understood and where appropriate action was so promptly taken. He himself may well have been in part responsible for this, having brought to Baltimore the current experience in Boston. The assimilation of this experience, however, was dependent upon an unusually alert and studious professional group. Where else at that particular time could he have been cared for by such physicians and surgeons?—men who, without exception, as the years went by became, like himself, beyond measure distinguished.

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# A FOLLOW-UP ON THE JACKSTONE CALCULI REMOVED BY DR. HALSTED

RICHARD H. FOLLIS, JR., M.D., BALTIMORE, MD.

ON Dec. 9, 1891, an 84-year-old white man presented himself at the Johns Hopkins Hospital and was admitted because of the complaint of painful and frequent micturition. Physical examination revealed a distended bladder and an enlarged prostate. As it was considered inadvisable to explore the bladder with an instrument, two days later Dr. Halsted performed a suprapubic cystotomy.

"An incision into the bladder large enough to admit 2 fingers was made and five calculi were extracted with the fingers. These calculi were lying free in the pouch behind the prostate. They were light in weight and light grayish brown in color. The largest calculus, the one first extracted, resembled so strikingly the ordinary Jackstone that the five stones have always been referred to as *Jackstone* calculi. The perfect one has six prongs of about equal length, joining a small hub at the center: These prongs are so inserted into the hub that each one is at right angles to all of the others except the one which is in a direct line with it. Each prong is bifid at its tip. The smaller stones are less perfect but they appear to be all of the same type."[1]

Incidentally, the original photograph of these calculi which I have shown you was made by the James S. Cummins Studio, 106 N. Charles Street. One wonders if Dr. Halsted took them there himself.

Now we shall have to skip half a century in our story. Upon returning to Johns Hopkins in 1946, I came upon some calculi, one of which resembled a perfect jack, in one of our gross teaching rooms. Being certain that they had not been there before the war and remembering the vivid impression that those described by Dr. Halsted had made upon me years before, I wondered if by chance these could be the ones he had described. When his paper was consulted, it was quite obvious that they indeed were.

Subsequently, it was learned that the calculi had been found in Dr. MacCallum's effects and were placed in the teaching room. How he came by them is unknown.

More intriguing, however, in rereading Dr. Halsted's account of his calculi and his discussion of somewhat similar ones in the Hunterian Museum of which chemical analysis had been made, we found the statement:

"Unfortunately our *Jackstone calculi* were so carefully laid away by someone before a chemical analysis could be made that we cannot find them."

After commenting on certain other aspects of such calculi he closes his report:

"It is greatly to be regretted that an examination of our calculi could not have been made; but I still believe that some day they will be found."

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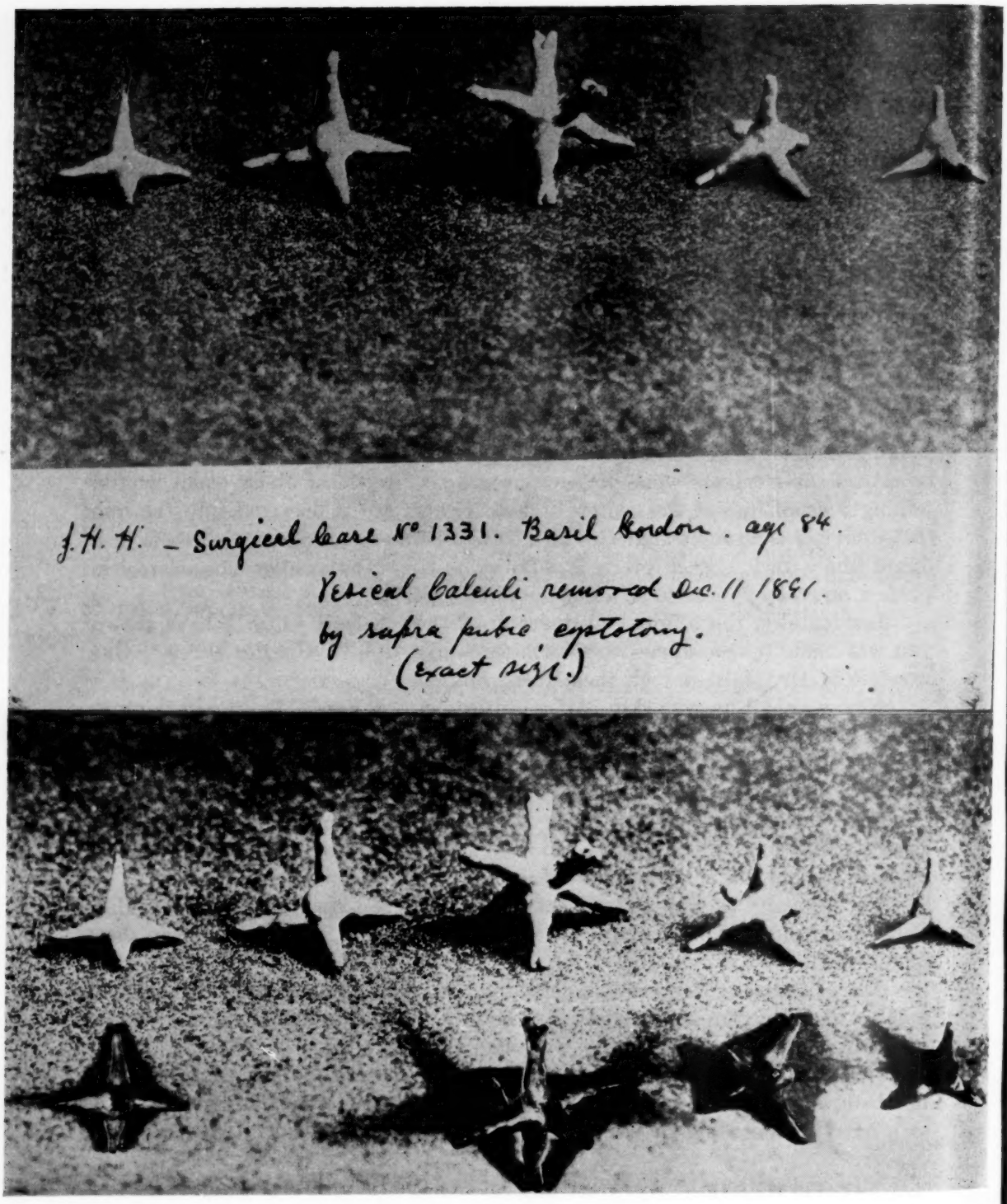
Read at the Centenary Celebration of the birth of William Stewart Halsted, Baltimore, Md., Feb. 6-9, 1952.

*A.*




*B.*

Fig. 1.—*A*, Photograph of Dr. Halsted's original copy of picture of Jackstone calculi with notation beneath.

*B*, Same picture upon which have been placed the original calculi except the second which was taken for analysis.

I shall not go into the details of the methods of chemical analysis. The calculus which was sacrificed proved to be composed almost entirely of calcium oxalate.

I feel privileged in being able to have fulfilled Dr. Halsted's belief that the calculi some day would be found and to close for him this morning, sixty years later, the case of the *Jackstone calculi.*

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# THE FRUITION OF HALSTED'S CONCEPT OF SURGICAL TRAINING

B. NOLAND CARTER, M.D., CINCINNATI, OHIO

THE resident system of training surgeons, which Dr. Halsted inaugurated at the Johns Hopkins Hospital in 1889, was not only the first program of its kind in America, but was unique in its primary purpose, and remained so for many years.

Halsted wished, above all, to establish a school of surgery which would eventually disseminate throughout the surgical world those principles and attributes which he considered sound and proper. In order to accomplish this practical and far-sighted purpose, he proposed to establish a training system which, in his own words, would produce "not only surgeons but surgeons of the highest type, men who will stimulate the first youth of our country to study surgery and to devote their energies and their lives to raising the standards of surgical science." His aim was to train *teachers,* and not merely competent operating surgeons. He, therefore, carefully selected as members of his resident staff men whom he considered usually gifted and promising and who, upon completion of their own training, would be eager to go out and teach.

The soundness of this concept can best be appreciated when one considers briefly the situation that existed in that period as regards the teaching of surgery and the opportunities for an adequate surgical training. A good teacher of surgery was a scarce commodity. For various reasons, it was difficult to attract well-known surgeons to chairs of surgery: first, because of the poor salaries attached; second, renowned surgeons carried on their work in hospitals which were usually isolated from university centers; and last, but perhaps most important, the more successful surgeon was not inclined to pass on to others his hard-earned skill which had been acquired under the most difficult conditions. Research and the practice of surgery were widely separated, and among surgeons there was little exchange of knowledge, as well as little intellectual stimulation. As far as the training of surgeons was concerned, there were few opportunities in hospitals for constructive teaching, the acquisition of an adequate training was a matter of personal will, and most surgeons were self-taught.

It would be most difficult to measure accurately the extent of Dr. Halsted's influence on the practice and teaching of surgery. However, it seems of interest to indicate at this time how well his fundamental purpose of training teachers of surgery has been fulfilled. To this end, a survey has been made, and is herewith presented, of his resident surgeons, his assistant resident surgeons, and the second generation of resident surgeons trained by *his* resident surgeons. The data for this survey have for the most part been obtained by personal communication, supplemented when necessary by reference to the various directories and journals.

The members of Dr. Halsted's resident staff, which usually consisted of three assistant resident surgeons and one resident surgeon, were most carefully selected. They were chosen on the basis of their ability and promise of future development, regardless of where they had received their education and train-

Read at the Centenary Celebration of the birth of William Stewart Halsted, Baltimore, Md., Feb. 6-9, 1952.

ing, although, as a matter of fact, the majority were medical graduates of Johns Hopkins University. The competition among this group for the coveted position of resident surgeon was keen, to say the least, and has even been described as "fierce and rigorous." There was no set length of time required for the attainment of this position, nor was there any orderly succession to it. The

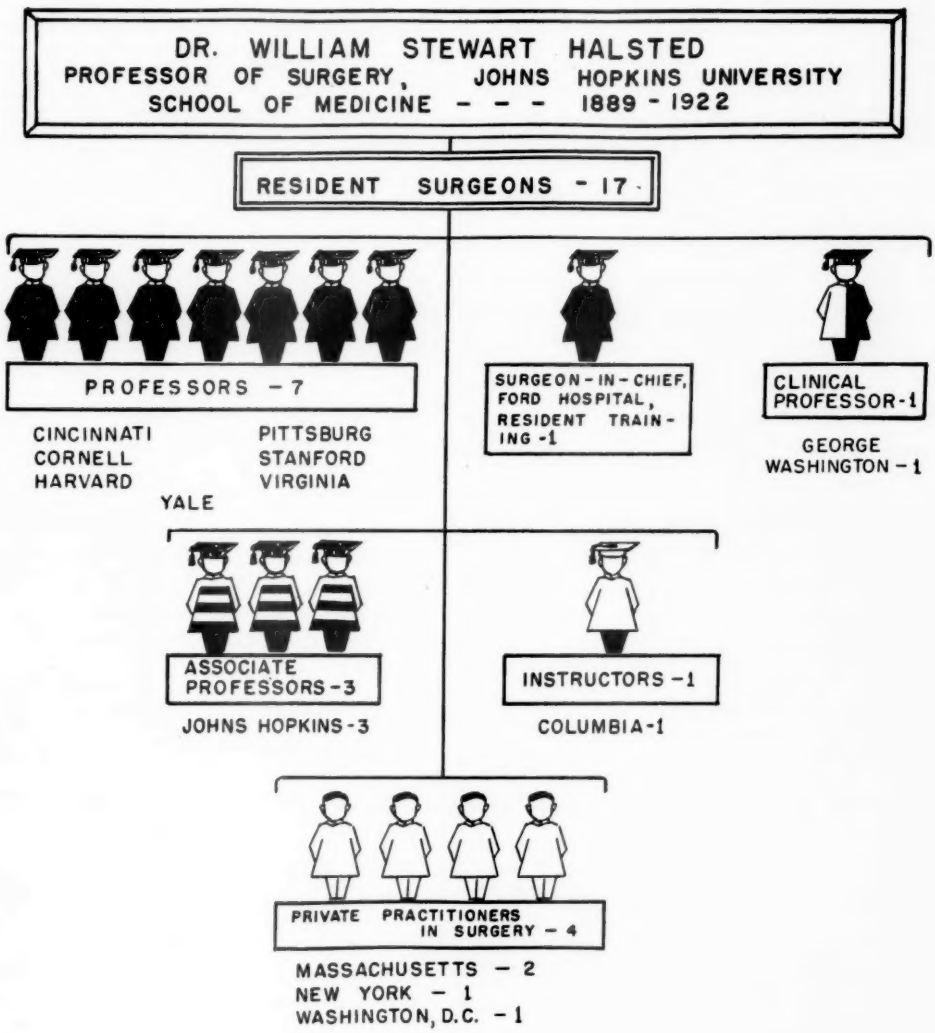


Fig. 1.

impression has been widespread that Halsted's resident surgeons served for many years on his resident staff, both as assistant resident and as resident. Actually, the figures are as follows:

2 men served 7 years each
4 served approximately 5½ years each
5 served approximately 4½ years each
4 served approximately 3½ years each, and
2 served approximately 1 year each

TABLE I. TEACHING RANK OF HALSTED'S RESIDENT SURGEONS

| | | |
|---|---|---|
| John W. Churchman | Professor of Surgery | Yale University |
| Harvey Cushing | Professor of Surgery | Harvard University |
| George J. Heuer | Professor of Surgery | University of Cincinnati, Cornell University |
| Emile Holman | Professor of Surgery | Stanford University |
| Robert T. Miller | Professor of Surgery | Pittsburgh University |
| Mont R. Reid | Professor of Surgery | University of Cincinnati |
| Stephen Watts | Professor of Surgery | University of Virginia |
| Roy D. McClure | Surgeon-in-Chief | Henry Ford Hospital, Detroit |
| James F. Mitchell | Professor of Clinical Surgery | George Washington University |
| Joseph C. Bloodgood | Associate Professor of Surgery | Johns Hopkins University |
| Walter Dandy | Associate Professor of Surgery | Johns Hopkins University |
| Richard Follis | Associate Professor of Surgery | Johns Hopkins University |
| Frederick Brockway | Instructor in Anatomy | Columbia University |

In his final selection of a man for the highly prized residency, Halsted stressed such qualifications as originality, capacity for independent thinking, a real interest in teaching others, and adequate technical skill. During the 33 years in which he directed his system of surgical training, he appointed 17 resident surgeons. The average length of time spent by most of the men as resident was between 2 and 2½ years; however, Bloodgood remained for 4½ years and Reid for 3½ years. Out of these 17 men came 7 Professors of Surgery, 1 Surgeon-in-Chief of a hospital, 1 Professor of Clinical Surgery, 3 Associate Professors of Surgery, 1 Instructor of Anatomy, and 4 private practitioners in surgery (see Figs. 1 and 2, and Table I).

Fifty-five men served only as assistant resident surgeons under Dr. Halsted. They remained on the resident staff for periods ranging from 1 to 3 years,

Fig 2.—Geographic distribution of 17 Halsted Resident Surgeons.

TABLE II. TEACHING RANK OF HALSTED'S ASSISTANT RESIDENT SURGEONS

| | | |
|---|---|---|
| William S. Baer | Professor of Orthopedic Surgery | Johns Hopkins University |
| Frederick H. Baetjer | Professor of Roentgenology | Johns Hopkins University |
| George Blumer | Professor of Medicine | Yale University |
| B. Noland Carter | Professor of Surgery | University of Cincinnati |
| Samuel J. Crowe | Professor of Otolaryngology | Johns Hopkins University |
| Willis D. Gatch | Professor of Surgery | University of Indiana |
| Emil Goetsch | Professor of Surgery | Long Island College of Medicine |
| Francis R. Hagner | Professor of Genito-Urinary Surgery | Washington University |
| Howard C. Naffziger | Professor of Surgery | University of California |
| Frederick L. Reichert | Professor of Surgery | Stanford University |
| William Sharpe | Professor of Neurosurgery | New York Polyclinic |
| Adrian S. Taylor | Professor of Surgery | Peking Union Medical College, China |
| Carl W. Waldron | Professor of Oral Surgery | University of Minnesota |
| John L. Yates | Professor of Surgery | Marquette University |
| Hugh H. Young | Professor of Urology | Johns Hopkins University |
| Edmond D. Clark | Clinical Professor of Surgery | University of Indiana |
| Malvern B. Clopton | Clinical Professor of Surgery | Washington University |
| Sidney M. Cone | Clinical Professor of Orthopedic Surgery | University of Maryland |
| Charles F. Nassau | Clinical Professor of Surgery | Jefferson Medical College, Philadelphia |
| Jerome P. Webster | Clinical Professor of Surgery | Columbia University |
| John A. C. Colston | Associate Professor of Urology | Johns Hopkins University |
| Warfield M. Firor | Associate Professor of Surgery | Johns Hopkins University |
| Henry Hun | Associate Professor of Surgery | Albany Medical College |
| Jay McClean | Associate Professor of Research Surgery | Ohio State University |
| William F. Rienhoff, Jr. | Associate Professor of Surgery | Johns Hopkins University |
| Berwick B. Lanier | Assistant Professor of Surgery | University of Maryland |
| Harry R. Slack, Jr. | Assistant Professor of Otolaryngology | Johns Hopkins University |
| Martin B. Tinker | Assistant Professor of Surgery | Cornell University |
| William Core Duffy | Assistant Clinical Professor of Surgery | Yale University |
| George R. Dunn | Assistant Clinical Professor of Surgery | University of Minnesota |
| Arthur O. Fisher | Assistant Clinical Professor of Surgery | Washington University |
| Joseph H. Kite | Assistant Clinical Professor of Orthopedic Surgery | Emory University |
| Edward M. Parker | Assistant Clinical Professor of Surgery | George Washington University |
| Camillus Bush | Instructor | University of California |
| Earnest G. Grey | Instructor | Johns Hopkins University |
| Charles H. Watt | Instructor | University of Georgia |
| Jean Paul Pratt | Instructor | University of California |
| George Walker | Associate Instructor | Johns Hopkins University |
| Karl Schlaepfer | Clinical Instructor | Marquette University |
| Lewis L. Redford | Associate Demonstrator | McGill University, Canada |
| Harold C. Parsons | Associate in Medicine | University of Toronto, Canada |

with an average of about 1½ years. Their caliber is indicated by their subsequent attainments, both academical and professional. As a group, they have exerted a profound influence on American surgery, and would seem to have clearly justified Halsted's evaluation of their worth in appointing them to his resident staff. In this group, special mention should be made of those men whom Dr. Halsted directed into subfields of surgery. Their outstanding accomplishments in their chosen fields speak for them. These were: Frederick Baetjer

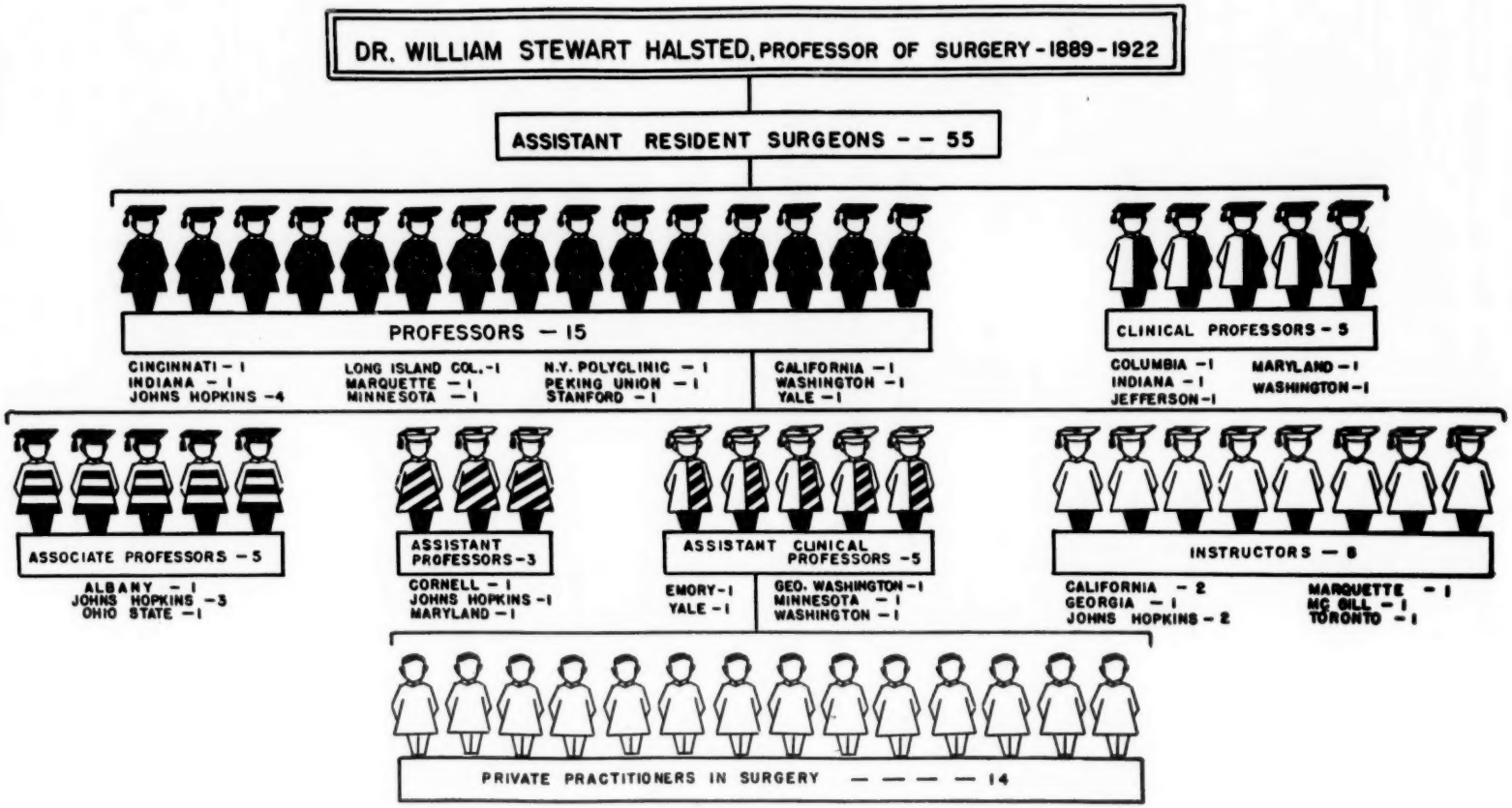
DR. WILLIAM STEWART HALSTED, PROFESSOR OF SURGERY - 1889 - 1922
ASSISTANT RESIDENT SURGEONS - - 55
PROFESSORS — 15
CINCINNATI - 1
INDIANA - 1
JOHNS HOPKINS - 4
LONG ISLAND COL. - 1
MARQUETTE - 1
MINNESOTA - 1
N.Y. POLYCLINIC - 1
PEKING UNION - 1
STANFORD - 1
CALIFORNIA - 1
WASHINGTON - 1
YALE - 1
CLINICAL PROFESSORS - 5
COLUMBIA - 1
INDIANA - 1
JEFFERSON - 1
MARYLAND - 1
WASHINGTON - 1
ASSOCIATE PROFESSORS - 5
ALBANY - 1
JOHNS HOPKINS - 3
OHIO STATE - 1
ASSISTANT PROFESSORS - 3
CORNELL - 1
JOHNS HOPKINS - 1
MARYLAND - 1
ASSISTANT CLINICAL PROFESSORS - 5
EMORY - 1
YALE - 1
GEO. WASHINGTON - 1
MINNESOTA - 1
WASHINGTON - 1
INSTRUCTORS — 8
CALIFORNIA - 2
GEORGIA - 1
JOHNS HOPKINS - 2
MARQUETTE - 1
MC GILL - 1
TORONTO - 1
PRIVATE PRACTITIONERS IN SURGERY — — — — 14

Fig. 3.

in roentgenology, William Baer in orthopedics, Hugh Young in urology, and Samuel Crowe in otolaryngology. Out of these 55 assistant resident surgeons developed 15 Professors, 5 Clinical Professors, 5 Associate Professors, 3 Assistant Professors, 5 Assistant Clinical Professors, 8 Instructors, and 14 private practitioners in surgery (see Figs. 3 and 4, and Table II).

Fig. 4.—Geographic distribution of 55 Halsted Assistant Resident Surgeons.

Going back to the 17 resident surgeons for the purpose of further tracing the Halstedian influence, 11 of these men in turn instituted resident training systems in surgery, similar to that of Halsted. From these systems have come a second generation of 166 resident surgeons trained in the Halsted tradition. From them have come also many more assistant residents, in whom the same tradition has been instilled, though no attempt has been made to survey this latter group. From this second generation of 166 resident surgeons have emerged 14 Professors, 8 Clinical Professors, 10 Associate Professors, 14 Clinical Associate Professors, 14 Assistant Professors, 11 Assistant Clinical Professors, 14 Instructors, and 81 private practitioners in surgery (see Figs. 5 and 6, and Table III).

When one summarizes numerically the results of Dr. Halsted's system of training surgeons, as related to his purpose of producing teachers, the following striking facts are evident. Among the group of 238 men comprising his resident surgeons, assistant resident surgeons, and the second generation of resident surgeons there were 37 Professors, 14 Clinical Professors, 18 Associate Professors, 14 Clinical Associate Professors, 17 Assistant Professors, 16 Clinical Assistant Professors, 23 Instructors, and 99 private practitioners in surgery, (see Figs. 7 and 8).

How well Dr. Halsted succeeded in his broad purpose of founding a school of surgery should be impressively evident from the study which has just been presented. It is also evident that in the fulfillment of his purpose, he succeeded

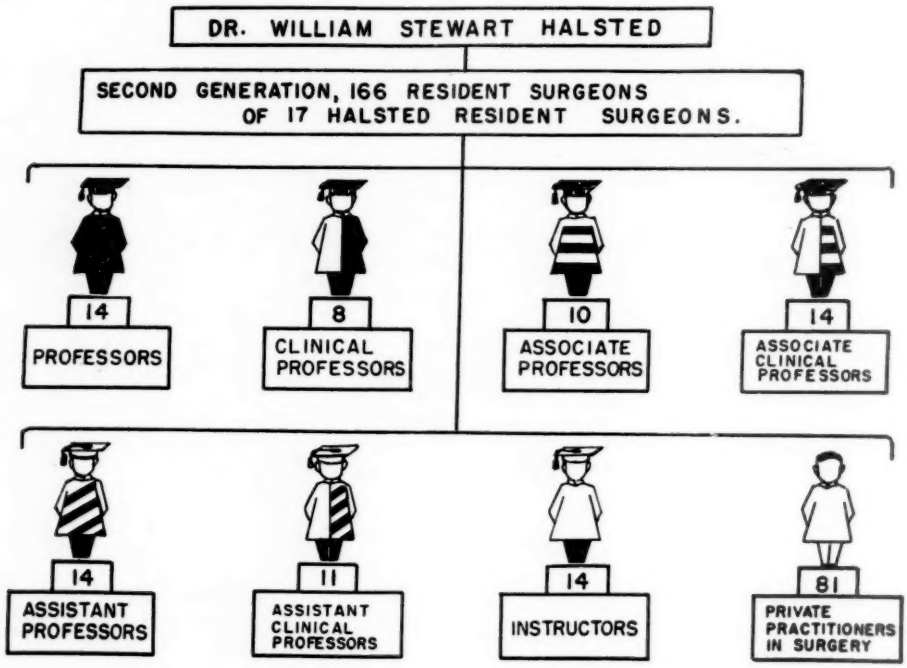


Fig. 5.

Fig. 6.—Geographic distribution of 166 second generation Resident Surgeons of 17 Halsted Resident Surgeons.

in training a group of teaching surgeons who were so imbued with his principles of thought and action, that they in turn are successfully handing down these principles to *their* students and assistants. It is indeed a rare occurrence for an individual to have his dream so completely fulfilled.

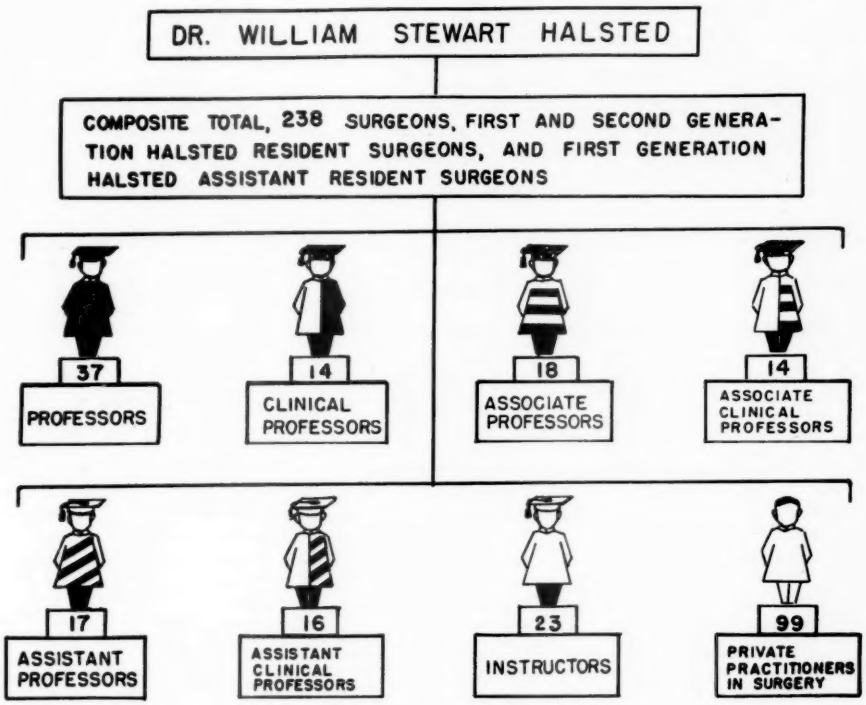


Fig. 7.

Fig. 8.—Composite total geographic distribution of 238 surgeons: first and second generation Halsted Resident Surgeons and first generation Halsted Assistant Resident Surgeons.

TABLE III. TEACHING RANK OF THE RESIDENT SURGEONS OF HALSTED'S RESIDENT SURGEONS

| | | |
|---|---|---|
| Winchell M. Craig | Professor of Neurosurgery | University of Minnesota |
| Edward J. Cumings | Professor of Ophthalmology | University of Maryland |
| Clarence E. Bird | Professor of Surgery | University of Louisville |
| Elliot C. Cutler | Professor of Surgery | Harvard University |
| Daniel C. Elkin | Professor of Surgery | Emory University |
| Wm. DeW. Andrus | Professor of Surgery | Cornell University |
| H. Glenn Bell | Professor of Surgery | University of Californa |
| Frank Glenn | Professor of Surgery | Cornell University |
| Edgar J. Poth | Professor of Surgery | University of Texas |
| Fred Sanderson | Professor of Surgery | Georgetown University |
| John V. Goode | Professor of Surgery | Southwestern University |
| Clyde Deming | Professor of Urology | Yale University |
| Isaac A. Bigger | Professor of Surgery | University of Virginia |
| John H. Neff | Professor of Urology | University of Virginia |
| Edward A. Kitlowski | Clinical Professor of Plastic Surgery | Johns Hopkins University |
| Arthur N. Owens | Clinical Professor of Plastic Surgery | Tulane University |
| Francis C. Newton | Clinical Professor of Surgery | Harvard University |
| Bronson S. Ray | Clinical Professor of Surgery | Cornell University |
| Robert A. Scarborough | Clinical Professor of Surgery | Stanford University |
| Richard F. Rand | Clinical Professor of Obstetrics and Gynecology | Yale University |
| Paul R. Sieber | Clinical Professor of Surgery | University of Pittsburgh |
| William H. Goodwin | Clinical Professor of Surgery | University of Virginia |
| Barton McSwain | Associate Professor of Surgery | Vanderbilt University |
| Franc D. Ingraham | Associate Professor of Surgery | Harvard University |
| William A. Barnes | Associate Professor of Surgery | Cornell University |
| Charles E. Troland | Associate Professor of Neurological Surgery | Medical College of Virginia |
| Ralph F. Bowers | Associate Professor of Surgery | Cornell University |
| Eugene E. Cliffton | Associate Professor of Surgery | Cornell University |
| David Henry Poer | Associate Professor of Surgery | Emory University |
| M. M. Zinninger | Associate Professor of Surgery | University of Cincinnati |
| Paul Kiernan | Associate Professor of Surgery | Georgetown University |
| Henry Hollenberg | Associate Professor of Surgery | University of Arkansas |
| Edwin I. Bartlett | Associate Clinical Professor of Surgery | University of California |
| Courtney C. Bishop | Associate Clinical Professor of Surgery | Yale University |
| Charles G. Child | Associate Clinical Professor of Surgery | Cornell University |
| Herbert Conway | Associate Clinical Professor of Plastic Surgery | Cornell University |
| William A. Cooper | Associate Clinical Professor of Surgery | Cornell University |
| John H. Eckel | Associate Clinical Professor of Surgery | Cornell University |
| Cranston W. Holman | Associate Clinical Professor of Surgery | Cornell University |
| Samuel W. Moore | Associate Clinical Professor of Surgery | Cornell University |
| William F. Nickel | Associate Clinical Professor of Surgery | Cornell University |
| E. Cooper Person, Jr. | Associate Clinical Professor of Surgery | Cornell University |
| Paul W. Sutton | Associate Clinical Professor of Surgery | University of Cincinnati |
| Leonard G. Dobson | Associate Clinical Professor of Surgery | Stanford University |
| Frank Gerbode | Associate Clinical Professor of Surgery | Stanford University |
| Nelson J. Howard | Associate Clinical Professor of Surgery | Stanford University |

TABLE III—CONT'D

| | | |
|---|---|---|
| George S. Gavatos | Assistant Professor of Surgery | University of Maryland |
| Dudley P. Bowe | Assistant Professor of Surgery | University of Maryland |
| Arthur J. McClean | Assistant Professor of Pathology | University of Oregon |
| Arthur I. Chenoweth | Assistant Professor of Surgery | University of Alabama |
| Richard Meagher | Assistant Professor of Surgery | Cornell University |
| Frank J. Otenasek | Assistant Professor of Neurological Surgery | Johns Hopkins University |
| Victor Richards | Assistant Professor of Surgery | Stanford University |
| William A. Altemeier | Assistant Professor of Surgery | University of Cincinnati |
| Paul I. Hoxworth | Assistant Professor of Surgery | University of Cincinnati |
| C. Marshall Lee, Jr. | Assistant Professor of Surgery | University of Cincinnati |
| Edward J. McGrath | Assistant Professor of Surgery | University of Cincinnati |
| Vinton E. Siler | Assistant Professor of Surgery | University of Cincinnati |
| Jean M. Stevenson | Assistant Professor of Surgery | University of Cincinnati |
| Francis M. Woods | Assistant Professor of Surgery | University of Cincinnati |
| Clarence L. Cohn | Assistant Clinical Professor of Surgery | Johns Hopkins University |
| George A. Stewart | Assistant Clinical Professor of Surgery | Johns Hopkins University |
| John E. Scarff | Assistant Clinical Professor of Surgery Neurosurgery | Columbia University |
| Arthur D. Console | Assistant Clinical Professor of Surgery | Cornell University |
| James A. Dingwall | Assistant Clinical Professor of Surgery | Cornell University |
| Harold Genvert | Assistant Clinical Professor of Surgery | Cornell University |
| John P. West, Jr. | Assistant Clinical Professor of Surgery | Cornell University |
| Joseph H. Boyes | Assistant Clinical Professor of Surgery | University of Southern California |
| Lot D. Howard, Jr. | Assistant Clinical Professor of Surgery | Stanford University |
| Frederic P. Shidler | Assistant Clinical Professor of Surgery | Stanford University |
| Richard Jackson | Assistant Clinical Professor of Surgery | George Washington University |
| Thomas Bowyer | Instructor in Gynecology | University of Maryland |
| Firmadge K. Nichols | Instructor in Gynecology | Johns Hopkins University |
| John H. Powers | Instructor in Surgery | Columbia University |
| Edward W. Douglas | Instructor in Surgery | University of Rochester |
| Charles Neumann | Instructor in Surgery | Cornell University |
| John B. Ogilvie | Instructor in Surgery | Cornell University |
| Edward W. Doherty | Instructor in Surgery | Stanford University |
| John G. Hemming, Jr. | Instructor in Surgery | University of Colorado |
| E. V. Ferguson | Instructor in Surgery | University of Cincinnati |
| James Mack | Instructor in Surgery | University of Cincinnati |
| Marcus Magnussen | Instructor in Surgery | University of Cincinnati |
| B. Z. Cashman | Instructor in Surgery | University of Pittsburgh |
| John W. Chambers | Instructor in Neurological Surgery | Johns Hopkins University |
| Bernard Maisel | Instructor in Surgery | Cornell University |

For the most part, the first generation of Halstedians has played its part in a well-conceived plan, and has played it exceedingly well. Were the Professor alive today, the group which would intrigue him most would be those 166 men who represent the second generation of resident surgeons. For they are the ones to whom it is now given to carry his torch and to create a third and larger generation of surgeons, who will also be imbued with the principles of their remarkable progenitor, William S. Halsted.

# HALSTED'S INFLUENCE UPON BRITISH SURGERY

DAVID H. PATEY, M.S., F.R.C.S.,* LONDON, ENGLAND

I AM very conscious of the honor and responsibility in speaking today on this historic occasion and before this distinguished audience as a representative of British surgery. Even one much more worthy of this honor than myself could hardly have failed on such an occasion of feelings of humility and reverence: reverence for the great surgeon we are commemorating, and for the great hospital and medical school in which he worked; and reverence also—and here I as an Englishman can speak more freely than you—for your and his great country on which, under God, so many of the hopes of mankind today rest.

It is my privilege to talk on the influence of Halsted on British surgery. The first and greatest influence is the unconscious influence. Just as in our common language what we say and how we say it are profoundly influenced by Shakespeare, John Bunyan, and the Bible, whether we are conscious of the fact or not, so in surgery what we do and how we do it are profoundly influenced by Halsted, again whether we are conscious of the fact or not. Halsted is an integral part of our make-up. Those of you who are amateurs of the beautiful, expressive, but difficult language of France, will know that the French translation of "to be an integral part of" is "faire corps avec quelque chose"—"to be part of the body of something." And Halsted is part of the body of British surgery. And that British surgery is fully aware of this relation is shown by the fact that the surgical section of the Royal Society of Medicine, also this year, independently, decided to hold in London a special Halsted centenary meeting, which Dr. Blalock and Dr. Crowe have graciously agreed to honor.

All that remains, therefore, is to consider first, what is the essence of the contribution of Halsted that has been such a tremendous influence, and second, how it was transmitted to England. On the first question I cannot attempt to emulate what has already been said so beautifully in the past by Rudolph Matas, and by René Leriche, and also by the distinguished speakers today. I will, therefore, add only this: The contribution of Halsted to surgery is in the direct high line of Hunter and Lister. All three saw and determined that the future advance of surgery lay along the path of experimental biology; all three developed a way of thinking and technical methods for illuminating and exploring this path. It was due to Halsted that the operation and the operating theater were raised to their rightful position, scientifically, of key points in an experimental biological process. Halsted's technical contribution was to discover the importance of delicate tissue craftsmanship and meticulous hemostasis as instruments of scientific surgery. It is on the scientific approach and technical methods of Hunter, Lister, and Halsted that modern surgery has been built up.

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Read at the Centenary Celebration of the birth of William Stewart Halsted, Baltimore, Md., Feb. 6-9, 1952.

*Mr. Patey represented The Royal Society of Medicine and The Royal College of Surgeons.

On the question of the ways in which Halsted's influence was transmitted to British surgery, I have already referred to the process of unconscious diffusion by which so much in the realm of ideas is transmitted. Of more specific ways, we all know that Halsted's main personal reciprocal relations were with the great German schools of surgery, with Kocher in Switzerland, and with René Leriche in France. But I think it can be fairly claimed that Halsted's relations with a British surgeon, Victor Horsley, represent the ideal of knightly chivalry between professional colleagues. You will all be familiar with Mont Reid's story in his account of Halsted's Friday morning clinics. You will remember how Halsted and Horsley both independently discovered that microscopic sections of hypertrophying thyroid after partial thyroidectomy in dogs were so unlike normal thyroid that Kocher and other great thyroid surgeons failed to recognize them for thyroid; and how both Halsted and Horsley did all they could to give credit to the other, even on one occasion to the extent of holding up the publication of a paper. The story illustrates the same point as Leriche's story which he gives in his book* of how Halsted suppressed his part in Dandy's discovery of ventriculography. When great minds and great men meet, the pettiness of life just falls aside.

But the personal channel for the communication of Halsted's ideas to England was largely through his pupils, and, in particular, Harvey Cushing. And the great fertilizing influence of Halsted is continuing today through his professional descendants in subsequent generations, among whom none is more worthy of mention than the present holder of Halsted's chair here in the Johns Hopkins.

Since sincerity and frankness are the test of friendship, I would be false to that friendship if I led you to believe that what I have said so far represents the whole of the attitude of British surgery to Halsted. Great men and great causes excite many reactions among men ranging from idolatry to debunking. And if among visitors from England to your shores you get the occasional debunker, I know you will regard this merely as one more proof of the infinite variety of the human mind.

Finally, great men have a significance in the realm of the spirit which cuts across all frontiers. Men who, like Halsted, undertake high tasks are in some sense contending for the honor of the human spirit.

As the *London Times* said of the recent British Mount Everest expedition: "In an age whose heart is dulled by the levelling of standards to the mediocre measure of the mass, here is an aristocracy that all can admire without envy."

I will conclude with the motto of our Royal Air Force—"Per Ardua ad Astra." Halsted's mind worked through patient and arduous detail, but it was fixed on the stars.

*La Philosophie de la Chirurgie.

# THE STORY OF THE DISCOVERY OF DENTAL ANESTHESIA BY NERVE BLOCKING

## Achievements of William Stewart Halsted

Rudolph Matas, M.D., New Orleans, La.

OF THE many innovations and discoveries that Dr. Halsted contributed to the progress of surgery, probably none is of more universal application and has been more thankfully received by the suffering world than his discovery of the method of nerve blocking for the complete control and suppression of dental pain, and in none is his priority more clearly established.

In this paper I will omit all discussion that relates to the well-known facts that pertain to Dr. Halsted's personality, to his premedical career, and to his early, brilliant, and unsurpassed surgical activities in New York up to the time of the discovery of cocaine. This discovery revolutionized the practice of ophthalmology as revealed by Koller's experience, told at the now historic Congress of Ophthalmology at Heidelberg in 1884.

This narrative is especially related to Dr. Halsted's achievements in the discovery of the application of cocaine to general surgery by the introduction of the drug into the tissues with the hypodermic syringe, not by simple external or epidermal application, but by the deep penetration of the injecting needle into the nerves themselves. He thus discovered the control and suppression of pain by the introduction of the analgesic fluid, cocaine, into the nerves controlling the sensibilities of the nerves of the teeth. This discovery laid the foundation for the practice of nerve blocking which has become, in its various modifications, the most beneficient and widely applicable of the discoveries which are so inseparably attached to his name.

I will begin with a summary of the main facts pertinent to the story of this achievement concluding with the grateful manifestations of the dental profession that came to him with rewarding glory toward the close of his unsurpassed career of surgical invention, crowning the latter years of his life with the recognition that he was one of the greatest contributors to the abolition of pain in dentistry.

A letter from Dr. H. D. Noyes, one of the delegates to the Heidelberg Congress, giving a full account of the proceedings of the congress, was published in the New York Medical Record of Oct. 11, 1884. This date may be taken as that of the first news of Koller's discovery to reach the medical profession of this country with sufficient detail to justify experimentation with the new anesthetic.*

In a survey of the literature which I undertook in 1899,[1] I had collected references and texts to over 60 published papers, essays, monographs, manuals,

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Read by Dr. Emile Holman at the Halsted Centenary Celebration of the birth of William Stewart Halsted, Baltimore, Md., Feb. 7, 1952.

*This summary has been extracted from my address: "Local and Regional Anesthesia: A Retrospect and Prospect," delivered before the American Society of Regional Anesthesia at the New York Academy of Medicine, Feb. 27, 1934, Am. J. Surg. 25: 189-196; 362-379, July and August, 1934. Included in my numerous papers regarding Dr. Halsted's priority is my very extensive correspondence with Dr. H. C. Bumpus of the Mayo Clinic, in 1931, in which I criticized his omission of Dr. Halsted's name *in toto* from the list of American pioneers in local and regional anesthesia in his address "History of Regional Anesthesia in Urology," J.A.M.A. 96: 83-88, 1931.

etc., which had appeared in the United States and Canada from October, 1884, to the end of 1885.

The workers who were experimenting with hypodermic or subcutaneous injections of cocaine in the fall of 1884, with different shades of purpose, may be compared to a rifle team engaged in a competitive shooting match. Many of these, firing practically at the same time, hit the target (local anesthesia), but very few hit the bull's-eye (neuroregional anesthesia). It is only fair, under these circumstances, that the one who hit the bull's-eye, not once, but often, regularly, and unerringly, showing that he was the expert in the crowd, should be adjudged the winner of the prize. And so it seems to me, in scanning the literature of that period, that Halsted and Hall were entitled to the award of priority above all their contemporaries for the first and most unequivocal demonstration of the principle of regional anesthesia and nerve blocking method.

It appears to me indubitably clear that the first premeditated use of cocaine on the principle of nerve blocking (by intraneural injection) for surgical anesthesia was performed by W. S. Halsted and R. J. Hall early in November of 1884. In their experiments and operations, as described by Hall in a letter published in the *New York Medical Journal* of Dec. 6, 1884, it appears that a lipoma of the supraorbital region was removed painlessly by Halsted after he had injected the supraorbital nerve at the supraorbital notch, that Hall had painless dentistry done on his front teeth by injecting the infraorbital nerve in the canal, that Hall and a medical student had their lower teeth completely anesthetized by an injection into the inferior dental nerve (given by Halsted) at its entrance into the canal, and that these experiences on the dental nerve were successfully repeated in a number of cases in which they were tried by Halsted and Hall. Later, Dr. E. H. Raymond, a dentist, reported to the New York Odontological Society (Jan. 20, 1885) that early in December, 1884, he had induced Dr. Woodbury, who was suffering with a very sensitive cavity in a molar which was to be filled, to test the value of neuroregional anesthesia with cocaine. Dr. Woodbury went to Dr. Halsted's office where Halsted injected 13 minims of a 4 per cent solution into the inferior dental nerve at its entrance into the inferior dental canal. Complete anesthesia of the gums was obtained in eight minutes and Raymond was able to use the dental engine with his patient perfectly free from pain.[2]

In another case referred to him by Dr. McBride, Dr. Halsted[3] performed Paravicini's neurectomy for neuralgia of the inferior dental nerve. The operation was performed in the winter of 1884-1885 in his own bedroom with the assistance of Dr. Richard Hall and Dr. Frank Hartley. He cocainized the nerve at its entrance into the inferior dental canal just as he had done with Hall, Woodbury, and others. After exposing the nerve he clamped and excised a section of it. A complicating hemorrhage from the dental artery left an indelible recollection of this operation.

On April 30, 1885, Dr. Charles F. Stillman reported to the New York Orthopedic Society[4] a case of congenital talipes on which he had operated by "brisement forcé," after Dr. Halsted had completely anesthetized the foot by injecting the postibial nerves at the ankle.

In reporting this case, Dr. Stillman referred to the fact that "though the honor of the discovery of cocaine as a local anesthetic belonged to Germany, yet to this country and to New York City was due the credit of demonstrating that perfect anesthesia could be obtained by injecting the trunk of the nerve that supplied the part affected."

Many years later I learned through a personal letter from Dr. Halsted that, among other operations performed in the winter of 1884-1885, he had freed the cords and nerves of the brachial plexus imbedded in a cicatricial mass after injection of the roots of the plexus in the neck. He had performed this operation in a large tent on the grounds of Bellevue Hospital, "having found it impossible to carry out antiseptic precautions in the general amphitheatre at Bellevue where numerous anti-Lister surgeons dominated and predominated."[5]

In 1900, in preparing a report of my own work on local and regional anesthesia from 1885 to 1900, I undertook a general survey of the world literature available on the subject up to that time.[1, 6] As a result of this very extensive and laborious inquiry, I came to the conclusion that Halsted and Hall were entitled to recognition as the first to discover and apply the principles of nerve blocking for regional anesthesia.

In 1920, twenty years later, I again reviewed the literature of 1884-1885 with special reference to the chronology of the contributions which dealt with the neuroregional method of nerve blocking. This was done at the request of the late Dr. C. Edmund Kells[7] of New Orleans, chairman of a committee appointed by the American National Dental Association to investigate the origin and author of the method of regional anesthesia by nerve blocking which had become of such inestimable value in dentistry and oral surgery.

This second survey fully confirmed my conclusion of 1900 in giving unequivocal precedence to the work of Halsted and Hall. The only difference between these two reports was that in 1920 I had accumulated more material to prove Halsted's priority. I realized that heretofore I had not done him adequate justice as the leader and prime mover of the cocaine investigation which he had started in 1884-1885 which had definitely established the principle of nerve blocking by regional anesthesia.

In this second report I had the advantage of Halsted's living testimony, which I had not had in my report of 1900 when I did not know him personally and depended exclusively on the literature extant at that time for my conclusions. In 1920, in reply to queries from Dr. Kells and myself (March 20, 1920),[3, 5, 8] Halsted showed how quickly and actively he had seized upon the news of Koller's discovery to begin his pioneer work.

He wrote: "Within a week, or two at most, of the arrival of Koller's first paper announcing the effect of cocaine on the conjunctiva and cornea, we began active experimentation with this drug, hoping that it might prove of use in general surgery."

By "we" he meant himself as chief of the outdoor clinic of Roosevelt Hospital, Dr. Richard Hall his first assistant,* Dr. Frank Hartley, his second,

*Dr. Hall, a close friend and co-worker of Dr. Halsted, retired early to Santa Barbara, California, where he died in 1900, prematurely ending a brilliant career of great promise. See also, Halsted.[3]

Dr. Frank Markoe, his third, and Dr. George Brewer, Dr. Charles Powers, Dr. George Woolsey and Dr. Lucius Hotchkiss additional members of his staff.

"In addition," he says, "twenty-five to thirty students of the College of Physicians and Surgeons (Columbia), who were registered with me as their preceptor, were enlisted in the cocaine investigation. At the evening quizzes we began our injections into nerves, almost all the accessible nerves being tested, inferior dental with the rest." (See Supplement.)

As a result of my second inquiry into the evidence regarding priority, in 1920, I was able to submit a detailed report to the Dental Committee which conclusively proved: (1) that Dr. Halsted had forestalled all his contemporaries in the recognition of the principle of regional anesthesia by nerve blocking and had been the first to demonstrate its practical value in dental and oral surgery by many well-planned and successful demonstrations, (2) that apart from the nerves of the face and jaws he had been the first to apply the same principle to the anesthesia of the extremities (brachial plexus block, posterior tibial block [in operating for clubfoot], etc.), (3) that Halsted was the first to demonstrate the superiority of *intracutaneous* infiltration as compared with *hypodermal* injections in obtaining anesthesia of the skin (subsequently confirmed by Corning, Reclus, Schleich, Braun and others), (4) that he was the first to demonstrate in surgery the anesthetic effects of pure water and of very dilute solutions of cocaine by infiltration and massive edematization of the skin and subcutaneous area, under pressure, thus greatly reducing the toxicity of cocaine. These two last discoveries which have been attributed to Schleich (1888) had long been antedated by Halsted (1885).[3, 5, 9, 10] All these are fundamental discoveries which have profoundly influenced the practical evolution of local and regional anesthesia.

I have laid special stress upon Dr. Halsted's priority in regional anesthesia in these four procedures and especially in nerve blocking, because it is upon the principle of nerve blocking that the whole superstructure of neuroregional anesthesia is based, including spinal, paravertebral, parasacral, caudal, and splanchnic, which at present command all our attention.

Dr. Halsted's activities were suddenly cut short by his illness in the winter of 1885, just as he was preparing to complete a paper on the use and abuse of cocaine: "Suggested by its universally successful employment in more than 1,000 minor surgical operations."[9-11]

As is well known, he had unwittingly succumbed to the habit-forming properties of the very drug that he had been so industriously investigating and trying upon himself, quite innocent of any knowledge of its treacherous fascination. He alone, of the many who, unsuspecting, fell under the spell of cocaine in those early days, fortunately "succeeded in conquering it by superhuman strength and determination and came back restored to a splendid life of greater achievement."[12] But two years had to elapse before he was able to return to his normal activities and to his new responsibilities at Hopkins.

In the meantime, the surgical possibilities of the new anesthetic were being exploited in every country with new ideas, new methods, and new substitutes for the drug, particularly in Germany, France, and America, where local and regional anesthesia became the universal topic of surgical and medi-

cal discussion. During this period his own discoveries were soon buried under the great heaps of the periodical literature which had piled up during the fated years of his illness, and the methods which he had originated were dubbed with the names of investigators who had followed in his wake. Even his own pupils had no knowledge of his pioneer work or its fundamental importance.[13]

Halsted's horror of the drug contributed, no doubt, to his complete silence regarding his work in New York. He seldom or never referred to it until later years, when nearly two decades had passed away, when the present writer and others, historically bent, brought it out of the silence and oblivion which he himself had imposed upon it. It was not until April 1, 1922, barely six months before Dr. Halsted's death, that the fundamental importance and significance of his original discoveries in local and regional anesthesia were duly recognized by the American National Dental Association, which presented him with a beautifully designed gold medal and, in a great ovation, gave him full credit as the originator of the method of neuroregional anesthesia, or nerve blocking, especially as applied to dental practice. A full and very interesting account of this occasion will be found in Dr. Kells' book entitled *Three Score Years and Nine*.[14] The celebration amounted to a veritable apotheosis which was unique in its splendor and in its marvellous dedication to the great master. His letter to me following this occasion shows that Dr. Halsted, despite his known dislike of publicity, was human and not indifferent to the just praise of his fellows.[15] (See Supplement.)

SUPPLEMENT

It gives me special pleasure to be able to supplement these remarks with the authentic handwritten letters of Dr. Halsted to Dr. Kells and myself, which I have been fortunate in preserving. Copies of correspondence between Dr. Halsted, Dr. Kells, and myself follow. The original letters of this correspondence are being turned over for permanent preservation to the custody of the projected Halsted museum which is now being inaugurated at the Johns Hopkins Medical School.

Extracts from Halsted's letters confirm in his own words the facts of his discovery of nerve block anesthesia in dental practice, in this way definitely and irrefutably establishing his priority in the discovery of this marvellous contribution to the progress of dental practice.

1201 Eutaw Place,
Baltimore, Md.

March 29, 1920

Dr. C. Edmund Kells,
1237 Maison Blanche,
New Orleans, La.

Dear Dr. Kells:

I am very appreciative of Dr. Matas' kindness in telling you of our early work with cocaine and take pleasure in complying with your polite request for further details.

Dr. Richard Hall died many years ago in Los Angeles or Santa Barbara. Hall and I were studying in Vienna in 1879 and 1880, and later were intimately

associated in surgical work at the Roosevelt Hospital and in its Out-Patient Department, he being my first assistant, Frank Hartley the second and Frank Markoe the third. Dr. George Brewer of New York was also on my staff in this dispensary.

Within a week or two at most of the arrival in this country of Koller's first paper announcing the anaesthetic effect of cocaine on the conjunctiva we began active experimentation with this drug, hoping that it might prove of use in general surgery. By "we" I mean twenty-five or thirty students of the College of Physicians and Surgeons (Columbia), all having the B.A. degree, who registered with me as their preceptor. At the evening quizzes we began our injections into nerves, almost all of the accessible nerves being tested—the inferior dental with the rest.

Dr. Thomas A. McBride, a remarkably gifted physician with a large consulting practice, referred to me in the winter of 1884 and 1885 the wife of a wealthy and prominent citizen. She was a sufferer from trigeminal neuralgia, the pain being pretty well confined to the region supplied by the third branch. I decided to perform the Paravicini operation under local anaesthesia and designed a very broad clamp with which to seize the nerve. The purpose of the unusual breadth of the instrument was to insure the excision of a long piece of the inferior dental nerve.

The operation was performed in a well lighted bedroom of my house, the patient being assured that she could return home the same afternoon. I was assisted by Dr. Richard Hall and Dr. Frank Hartley. The nerve having been satisfactorily anaesthetized with a strong solution (4 per cent) of muriate of cocaine, was exposed and seized with the broad clamp without great difficulty. With a scissors I divided the nerve at the distal edge of the clamp. Then, guided only by the clamp and a finger, I made a snip with the scissors in the deep hole through the nerve at the central edge of the clamp. Thereupon there was a great gush of arterial blood—so great that I thought the internal maxillary artery must have been cut. The patient was in danger for a moment of being suffocated with the blood. By forcible packing with iodoform gauze the hemorrhage was stopped, but such pressure was made by the packing that I feared extensive sloughing of the soft parts would ensue. Confidently expecting a recurrence of the bleeding, I hastily summoned two trained nurses from the Presbyterian Hospital and had the patient put to bed. In a day or two she was transferred to the Presbyterian Hospital. There was no recurrence of the bleeding.

About the eighth day after operation and with considerable apprehension I cautiously removed the gauze packing. The patient recovered without a complication of any kind.

Strange to say, I did not know that Hall had reported this case until in 1914 at a meeting of the Deutsche Gesellschaft fur Chirurgie Professor Rehn, the President of the Congress, referred to it in the course of a discussion. From him I obtained the reference to Hall's paper; and I believe that Braun mentions it in his book on local anaesthesia.

Hall was appointed professor of anatomy at the Columbia Medical School, but on account of ill health he soon resigned. He became a successful and highly respected practitioner of surgery in Santa Barbara or Los Angeles. Dr. George Woolsey and Dr. Lucius Hotchkiss and other students of mine in New York could tell you something of our early experiments and operations with local anaesthesia.

I published at the time a few little paragraphs on the subject in the New York Medical Journal. One of these was to call attention to the fact that one could produce local anaesthesia with injections of water. We soon discovered that very dilute solutions of cocaine sufficed, and in the first years of the Johns Hopkins Hospital I made extensive use of local anaesthesia with these very mild solutions—as weak sometimes as one to ten thousand.

Already in 1884 I had noted the effect of anaemia on the anaesthetized parts, and emphasized the importance of distending the tissues until they were blanched. I made use of the constricting rubber bandage on the limbs and of heavy rubber rings on the fingers in order to intensify and prolong the action of the drug. When small veins were accidentally injected we observed the production of urticaria-like wheals. To Schleich, very properly, belongs the credit he has received inasmuch as my work, owing to ill health, was not published.

In the fall of 1885 I had a few amusing experiences with the dentists in Vienna. Having occasion to consult Dr. Thomas, a famous "American dentist," with spacious offices on the "Graben," I demonstrated to him the effects of injecting the inferior dental nerve. His first assistant was so impressed that he requested me to inject his nerve, and thereupon asked Dr. Thomas to pull one or two roots and fill a tooth. Professor Anton Wolfler, the first assistant of Billroth, published a brief note on the subject of cocaine anaesthesia in one of the Vienna morning papers based on a demonstration which I had given him a day or two before of our method of using it for surgical operations. Prior to this demonstration he had convinced himself by experimentation that cocaine would not produce anaesthesia except on the surface of mucous membranes—that it was useless when injected into the tissues.

I beg that you will pardon me for indulging in such a lengthy reminiscence. Will you give my kind regards to Dr. Matas.

Very truly yours,
(Signed)
W. S. Halsted

1201 Eutaw Place,
April 3rd., 1922

Dr. Rudolph Matas,
2255 St. Charles Ave.,
New Orleans, La.

My dear, dear Friend:

How can I ever find words to express my gratitude to you for this act of unparalled kindness—an act which has covered two years.

The celebration, as Dr. Kells will tell you, was a remarkable success. I am so thankful to have lived to take part in it. Not a wink of sleep did I get during the night of Saturday; I was too exhilirated for repose.

Once before in my life I was kept awake by great happiness; this was the night that I passed successfully the examination for Bellevue Hospital—1876. Then it was in contemplation of the future, now in reflection upon the good fortune which led to our friendship.

The reaction from this great joy seems to be setting in tonight, and my happiness is tinged with regret for the lost opportunities—for the time wasted from loss of health.

I am glad that you were unable to come on, for my cup of gratitude was already overflowing. I hope that we may meet in Washington at the coming Congress and that we are to have a contribution from you.

I was delighted with Dr. Kells and feel that it was wonderfully kind of him to take the long journey to Baltimore and to preside at the banquet.

Good night, dear Matas.

Ever affectionately yours,
(Signed) W. S. Halsted

## CONCLUDING REMARKS

Dr. Halsted died Sept. 7, 1922, thirty years ago, and with his death the surgical world lost one of its greatest thinkers and leaders.

His death meant a great personal loss to me. I first came to know him in 1903 when he operated upon me and relieved me of worry caused by what I had feared was an incurable lesion. He not only gave me the benefit of his great skill, but provided the hospitality of his home where I convalesced for some time, and where I enjoyed the privilege of his care and confidence, for which I remain his debtor for all time.

In the course of the years that followed, we carried on a more or less continuous correspondence which kept me in touch with his constantly growing achievements. It culminated in a warm and confidential friendship—short of familiarity—which continued until his death.

Though it was not my fortune to have been one of his pupils, my long and thorough acquaintance with his work would seem to justify the assumption that I may be rightfully considered as one of them, and it is for this reason I regret the more acutely my inability to join this distinguished company of his pupils, associates, and friends in rendering homage to his memory which has remained ever green in my affections and thoughts.

I rejoice, none the less, that the magnificent tribute which has been paid to his personality and achievements has served to preserve his image with the vividness of his most active days, but with an aura of glory that appears to be more resplendent since his death.

I rejoice also in the fact, revealed by the scientific contributions that have poured out in such profusion at this meeting, that the seed that he planted in the soil of this great institution has fructified and is now yielding a harvest of knowledge and invention that not even he, the Master, could ever have foreseen as a reality such as we are contemplating here today.

My thoughts and best wishes are now directed to Dr. Halsted's successor and his associates, for the continuance of the ever-dazzling light of surgical knowledge that he left in their hands as his immortal legacy.

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# A TRIBUTE TO DR. HALSTED*

PROFESSOR RENÉ LERICHE, PARIS, FRANCE

I CAN hardly express the great pleasure you have given Mme. Leriche and myself in inviting us to commemorate with you the centennial of Dr. Halsted's birth.

It is indeed a great honor and I do not only appreciate that, in itself, but I must say that my joy surpasses everything.

That is because I have greatly loved and admired the one whose memory we are honoring today and I am happy to render him the tribute of my gratitude. It is also because I have had the great privilege of having been favored by his precious friendship.

I would like to tell you how that came about, for it was an unusual adventure: everything took place in three short days in the month of May, 1913.

At that happy time, I was a young surgeon full of curiosity. I was familiar with the surgeons of Germany and I was eager to get in contact with the great American surgeons of the time whose prestige Carrel had so often praised.

I really was very young, at least in appearance. I did not know very much English. It is true that I had my wife as interpreter, but I did not look forward to these meetings without a little apprehension.

Fortunately, I was guided by Dr. Alexis Carrel's friendship and by the kindness of Simon Flexner who had warmly welcomed me at the Rockefeller Institute. I was directed by them to the Johns Hopkins Hospital with the necessary recommendations.

Although I had the air of a novice, I was admirably received by Dr. Halsted, with a cordial simplicity that made us feel at ease right away. He took time to present me to all his staff, to show me around, to take me to visit his patients, and allowed me to watch him operate on a large iliofemoral aneurysm and a cancer of the breast.

I had been trained in the feats of surgery, I was schooled in the tradition of the ancients who considered the surgical performance as a magnificent act of prestidigitation, and I had no other ambition than to be a rapid surgeon, doing things daringly. I saw a man who was careful to avoid trauma, particular in the extreme, very sparing of the patient's blood, concerned about perfect tissue reconstruction. It was a dazzling revelation. In a split second I realized my error. A real revolution took place in my mind. From the first day I was convinced, and if one day I reduced postoperative morbidity, it was owing to Baltimorean reflections.

We spent a few hours in the laboratory. He was studying at that time, together with Mont Reid, the outcome of partially occluding arteries by a metallic band. While studying these specimens, we spoke at length about arterial surgery and I was surprised to find out all that he had studied on his own

*Delivered by Professor Leriche in French at the Centenary Celebration of the birth of William Stewart Halsted, Baltimore, Md., Feb. 6-9, 1952.

without ever having spoken about it. Was not this just the experience of Porta? He took me to Howell, to Abel, and caught in that experimental atmosphere, I understood, more than ever, how one should really work. I was too much inclined to consider that surgical work was purely clinical and technical. I had not learned my lesson from Ollier as I should have. It was at the Johns Hopkins Hospital that I did.

In three days I had gone through the experience of a great evolution. Obviously, that did not take root at that very moment, but when I returned home, I was a new man. I felt as if I had lived near the surgical heir of Claude Bernard.

I very often regretted not having stayed longer. But my means were limited. I had to leave for Chicago, Rochester, and Boston. At that time, it had not been fully understood that the real purpose of an educational tour is to learn thoroughly the teachings of one man. It was thought then that the only important thing was to meet the maximum number of surgeons in the minimum time. But the essential thing had been done for me. I had received the shock and in addition to that, I had gained a rare friendship, for this person was charming in his hours of relaxation. Simple, delightfully unaffected, cordial, easy-going, amused by everything, with that peculiar trait of never wanting to be photographed, for Mme. Leriche was never successful in obtaining his picture.

Toward the middle of 1914, he did me the honor of asking me to send him an assistant for a year. If I had dared, I would have proposed myself. I selected my friend Santy, who had just finished training and who had an innate understanding of the problems of surgery and the soul of an investigator. His ensuing brilliant career and especially his present immense experience in cardio-aortic surgery prove that the choice was good. Alas, war was to end all projects.

August, 1914. One of the first letters I received in the army, at the front at Vosges, was a letter from Dr. Halsted, about the twenty-fifth of August. He told me of his fervor for the French cause, the cause of the world, and of his sincere wishes. From that moment on, our correspondence was regular, more and more friendly from month to month. With a charming sense of understanding, he would tell me his hopes, those of his country, his regret at not being able to go to the front because of his health, his enthusiasm after the victory at Marne and over the resistance at Verdun. And so as to make me feel where the heart of America beat, he sent a subscription of *The New York Evening Post* to Mme. Leriche.

In each of his letters, he would kindly express concern over my fate and that of Dr. Santy; he kept me posted on news of our acquaintances; spoke to me about his research work, of his dahlias, the bulbs of which come from a place in Lyon and had found such suitable ground in the garden of his North Carolina home, of the million individual surgical dressings that he was having made to send to France, and, above all, about his faith in the triumph of justice. Occasionally, he would advise me of ways of treating infections. He recommended the continuous hot tub baths that I had occasion to try out and I know of patients with serious infections who owe their lives to the advice of faraway Halsted.

At the beginning of the war, censorship was not as rigorous as it was later on. We were able to take pictures and send them by mail. In January, 1915, I sent him some negatives that I had collected of wounds, of the trenches, and of the battlefield. He received them, but he would not keep them for himself. He exhibited them in a reception room of his club and I believe he even gave a short talk about them. Shortly after that, he sent me a generous check that he had collected for my wounded.

How could I help liking a man so thoughtful, so generous, so expressive?

The end of the war came and his letters were triumphant, full of an enthusiastic sympathy. I am happy to place in the hands of the Halsted Committee the letters of his that I was able to preserve amid the hazards of war. There are several missing, lost in the trips to and from the front line. The group of these remaining letters will suffice to show a side probably little known of the soul of Halsted.

Shortly after that, I received an invitation from the American Surgical Association for its meeting in Toronto. I always felt, although he never made the slightest reference to the subject, that I owed this honor to him. And so I returned to the United States in 1921. Of course, the first visit for my wife and myself was to Baltimore. We found the Professor just the same: hardly any older, pleasant and gracious as always. Again, pressed for time, I was able to stay for only three days. He reproached me in a friendly way for not staying as long as the first time. He could hardly believe that our first visit had not been any longer.

"I thought," he said, "that you had stayed so much longer then, our friendship seems so deep."

Those were delightful and fruitful days. I remember now an anecdote that might show you the importance that Halsted had in Baltimore. My wife had taken a taxi to Eutaw Place to show Miss Anna Stokes the English wording of my paper for the Toronto meeting, and the chauffeur, an old chauffeur, obstinately refused all remuneration, stating that friends of Dr. Halsted did not pay and that he was only too happy to take them, for Dr. Halsted had rendered so many services to everybody.

I would like to state briefly, before ending, my opinion of Dr. Halsted's place in the surgery of the world.

A quarter of a century after his death, he seems to me as one of the greatest surgeons of his time and of all times, because he is one of the chief founders of scientific surgery, because he created methods and procedures that have had a determining influence on the evolution of therapeutics, because he really founded a school, the disciples of which have perpetuated his influence in the world while being creators themselves.

Along with Lister and Ollier, he oriented surgery in the field of physiologic research, pure research, but without losing contact with the patient, thus tracing his way to scientific surgery.

There were many dominating minds in the field of surgery during the last half of the nineteenth century, but I know of no one who pointed out as he did the royal road to research. Billroth was an outstanding man who created a

great part of abdominal surgery and started a large and fruitful school, but he remained mostly in the domain of technical creation. Kocher was in the line of Halsted. Péan was in a class of his own, but he was above all an extraordinary virtuoso of unpublished surgery. I cannot think of anybody, with the exception of Ollier, who remained to the end an investigator.

Second, he created an operative method that became prevalent everywhere, and which permitted Cushing to do what he did and Alfred Blalock to create his admirable cardio-aortic surgery. Kocher and Terrier bent their efforts in the same directions, but no one codified as he did the procedure of atraumatic operation, the bloodless operation respectful of the means of life in general and of the life of tissues in particular. Very often during his lifetime it was thought that his detailed care and his slowness were excessive.

He himself used to say, "Perhaps I exaggerate, but it is necessary to do this in order that something endures."

He created the method of regional anesthesia by infiltration with his four principles: intradermal injection, the use of a solution very dilute in water, the blocking of nerves, and the role of reduced circulation which we practice nowadays by the vasoconstricting action of Adrenalin.

He was the first one to think of reinjecting lost blood arterially. He set the operative procedure that we all employ for cancer of the breast. Through his experimental studies he was a decisive influence in the surgery of thyroid, parathyroid, and thymus hypertrophy. He discovered the law of the grafting of glandular tissues. Finally, his contribution to vascular surgery is one of the most important up to modern times, and even in that field of surgery his influence is still evident here since Blalock is continuing Halsted.

Need I add that his monographs on goiter, on the ligation of the common iliac and of the left subclavian arteries are models of perception and precision?

Finally, he founded a school whose splendor has had no equal except in the brilliance of Billroth's students. To be perfectly truthful, each one of us, if he is sincere with himself, must recognize that he depends to a certain extent on the teachings of Halsted. Is it not for this reason that I have the honor of being here? I have this profound feeling and that is why I am happy to render to his memory the homage of my fervor and deep gratitude.

To this personal homage I would like to join that of the Academy of Surgery of Paris which associates itself wholeheartedly in the celebration of a great centennial.

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# HALSTED'S NEW YORK PERIOD

ALLEN O. WHIPPLE, M.D., PRINCETON, N. J.

IN PRESENTING the New York period of Dr. Halsted's career, a privilege which I deeply appreciate, I shall endeavor to bring out the significance of certain traditions and events which will explain the contrast of his life and personality in New York with that of his subsequent career.

As a prelude to his New York years it is pertinent to recount some of his earlier experiences as a student at Yale University.

In a letter to Dr. Welch, recounting his college days, Dr. Halsted wrote as follows: "Entered Yale without conditions in 1870 and graduated in 1874. Devoted myself solely to athletics in college."

In his sophomore year he played on his class baseball team, in his junior year he was a member of his class crew, shortstop on his class baseball team, and one of the twenty performers in the Yale gymnastic exhibition for the benefit for the Yale Navy. In his senior year he was on his class crew and captain of the varsity football team, or organization as it was then called. The most notable game of football in which he starred was a game Yale played against graduates of Eton. Yale won by the score of two goals to one, Halsted making the winning goal.

It is interesting to note in this letter to Dr. Welch the following statement: "In senior year purchased Gray's *Anatomy* and Dalton's *Physiology* and studied them with great interest; attended a few clinics at the Yale Medical School."

This was evidently the awakening of his interest in medicine. His decision to enter the College of Physicians and Surgeons may have been determined by his reading Dalton, who was Professor of Physiology at that College.

He came to New York, where his parents lived, in the fall of 1874, and matriculated in the College of Physicians and Surgeons. There he immediately demonstrated his interest and ability in his medical studies, especially in the subjects of anatomy and physiology. By the end of his second year he was an outstanding student and, against all precedent, he competed for the internship at the Bellevue Hospital in the beginning of his third year and received a surgical appointment. At the end of this year he stood among the first ten in the graduating class at the College, and in the final competitive examination for the one hundred dollar prize he stood first among the first ten men in the class.

During his internship at Bellevue he wrote: "I followed the medical work with quite as much interest as the surgical, making rounds with Janeway, Loomis, and Jacobi, attending autopsies and spending much time in the medical wards. I assisted Jacobi at his surgical operation (on the wards) which were very, very amusing."

Read at the Centenary Celebration of the birth of William Stewart Halsted, Baltimore, Md., Feb. 6-9, 1952.

In the spring of 1878 he competed in the examination for the position of House Physician at the New York Hospital and received the appointment.

He wrote: "Dr. Draper (the Attending Physician) was most friendly, inviting me frequently to his house, to musicales on Sunday afternoon and to dinner."

After this term at the New York Hospital, Halsted began his two years of study in the famous clinics abroad.

He wrote: "In the fall of 1878 I sailed for Europe. Sam Vander Poel, who had been in Germany for the previous six months, met me in Paris. The first morning we were invited to breakfast at Foyot's by friends of his from California, who nominally had been studying medicine in Paris for two years. We asked to be taken to L'Ecole de Medicine by these gay birds, who told us that they had been unable to discover it, considering it a hard day's work to go to the bank. Under guidance of this bevy we learned a few things about Montmartre and then took train for Vienna."

Once there he began to work with a vengeance. He took courses all day with men whose names are still famous, with Chiari in pathology, Fuchs and Arlt in diseases of the eye, Politzer and Gruber in diseases of the ear, Kaposi and Neumann in skin diseases, and with Pawlik in gynecology.

"But anatomy was my chief work" he wrote, "with Zukerkandl and Holl as my teachers. Attended clinics of Billroth and Braun. My work in embryology with Schenck was chiefly valuable because it led to friendly relations with Woelfler who was Billroth's first assistant, Mikulicz being another. What impressed me chiefly in Billroth's Clinic was the magnitude of the operations and the skill of Billroth and his assistants, particularly Mikulicz, and the great number of artery forceps used."

During the next year he studied in different cities in Germany under Kolliker, Stohr, Weigert, von Bergmann and with Volkmann for several weeks. It was Volkmann who was doing so much in those days in demonstrating the efficacy of Lister's doctrine of antisepsis. The last two surgeons he visited were Schede and Esmarch.

Early in September of 1880 he returned to New York. With his independent means, his splendid reputation before he went abroad, and the recognized fact that he had had such unusual training under world authorities in Austria an Germany, the gates of opportunity were opened to him on his return. He now began the most active period of his entire career. Sands, who was Professor of Anatomy and Surgery at the College of Physicians and Surgeons and Senior Attending Surgeon at the Roosevelt Hospital, invited him to be his associate in that hospital and allowed him to choose the cases he was most interested in and to use his recently acquired techniques. These techniques required an enlarged surgical armamentarium.

With Sands' permission Halsted ordered, among other things, a full dozen artery clamps, which seemed extravagant, for Sands asked, "What shall we do with so many?"

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In 1881 he was appointed surgeon to the Charity Hospital on Blackwell's Island, and the following year he became Chief Surgeon to the Emigrants' Hospital on Ward's Island. In 1883 he was appointed to the Surgical Staff of the Presbyterian Hospital, as well as to the summer service of the Chambers Street Hospital where William T. Bull was Chief Surgeon. This hospital had the most active traumatic and emergency service in the city. In 1884 he was appointed as Visiting Surgeon to the Bellevue Hospital. There he found the incidence of clean wound infection so deplorable that he asked the hospital authorities to put up a special operating room in the hospital yard away from any of the wards. They told him that this could not be done, but gave him permission to set up a special tent in the yard, to which Halsted's friends and his brother contributed.

A very interesting experience in his efforts to introduce aseptic techniques in the hospitals to which he had been appointed occurred in the Presbyterian Operating Room. He had been appointed to the service of Dr. Briddon who, as an older, English-trained surgeon, was totally unfamiliar with the principles of asepsis. In order to welcome Halsted and show him all the professional courtesies, he insisted, at first, on acting as first assistant, with the House Surgeon holding retractors. This House Surgeon's description of Halsted's first operation at that hospital, which I found recorded in one of the old bound volumes in the archives, is delightful in the veiled allusions to Halsted's efforts to keep Briddon's hands out of the wound. The operation was a fusion of a tuberculous knee joint. The House Surgeon describes Halsted's meticulous care in scrubbing his hands and arms and the leg of the patient as well as the draping of the operative field.

He closes his operative report with the following comment: "Dr. Halsted—sterile, Dr. Briddon—no comment, House Surgeon—?"

One of Dr. Halsted's first accomplishments after his appointment to the Roosevelt Hospital was his organizing the Surgical Outpatient Department or Clinic. This had the enthusiastic support of his Chief, Dr. Sands. It was here that he attracted a group of able and enthusiastic young surgeons, among them Richard Hall, Frank Hartley, George Brewer, Frank Markoe, and George Woolsey.

Hall and Halsted were known as the indefatigable twins because of their tireless energy, their knowledge of anatomy, their familiarity with the surgical literature, and their fearless operative skill. Working together, they had a telling part in demonstrating to New York surgeons the advantages of asepsis over antisepsis and the importance of minimizing tissue damage and of careful wound repair. They were pioneers in the therapy of appendicitis, or perityphlitis as it was then called.

Halsted stated in a letter to Dr. Welch: "Dr. Hackley (who had been a Visiting Physician at the New York Hospital when Halsted was a House Physician in 1878), recalling my eagerness to operate early in cases of perityphlitis, assigned to me in the early eighties two private patients with acute general peritonitis. Assisted by Richard Hall, I removed the appendix promptly

in each case, but with fatal result. These were the first appendectomies performed in New York.'' He refers to a later case of Hall's, the first successful appendectomy in this country.

I had the privilege, some twenty years ago, in reviewing the history of appendicitis in New York, of finding the record of this very case in the old bound volumes of the Roosevelt Hospital, which I rescued from the basement where they had been taken to be burned. Fortunately, it was in the summer when the main furnace was out. The report of the operation was written by George S. Huntington, then an intern at the Roosevelt, later the world-renowned Professor of Anatomy at Columbia. An 8-year-old boy was admitted to the Emergency Ward with what was thought to be a strangulated indirect inguinal hernia. Hall operated and found a gangrenous appendix in the hernial sac. He ligated the base of the appendix, removed it, and repaired the hernia. The boy made an uneventful convalescence.

In addition to all his hospital appointments and responsibilities, Dr. Halsted was Demonstrator of Anatomy at the College of Physicians and Surgeons and had the largest and most exclusive ''Quiz'' at the College, for it was made up of men with college degrees. I must digress for a moment to explain the quiz system which existed in New York at that time, and which was unique to medical training in that city. I am discussing it because of its effect upon New York surgeons and their hospital training, and because of the rather ironical part that Halsted played in it, for it was so contrary to his later philosophy in his residency program.

Prior to 1890 the curriculum at the Physicians and Surgeons consisted of lecture courses, with special emphasis on anatomy in the dissecting rooms and limited laboratory demonstrations in physiology in the preclinical years. The students had little if any opportunity to study disease in hospital wards. They could, but were not compelled to, attend the lectures in preparing for their examinations. It was largely a matter of ''lecture larnin' '' from lectures previously given.

Before 1880 the hospital internships were of twelve to sixteen months' duration and with no distinction between medical and surgical duties. With the increase in surgical work and the recognition of the importance of acute peritoneal infections as developed by Sands, Bull, and Markoe in the treatment of appendical, or perityphlitic, abscesses, surgical internships were started at the Bellevue, the New York, the Presbyterian, and the Roosevelt hospitals. Surgical anatomy became increasingly important in the competition for appointment to those internships. To prepare men for these examinations extracurricular quizzes were organized by some of the younger attending surgeons to give the medical students greater familiarity with the subjects that were being emphasized in the hospital competitive examinations. In the three years in the quiz most of the subjects in the medical school curriculum were reviewed, with special emphasis being placed on the idiosyncrasies of the hospital examiners. The students desiring hospital appointments were really exploited, for many of the quiz masters were on the College faculty and advised their students to pay more attention to their quiz work than to the lectures given in the College.

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It soon became evident that the students that had had the intensive coaching and discipline, most of it brute memorizing, in the quiz stood highest in the final hospital examinations, and secured the best appointments. But out of this quiz system, which was considered essential for anyone seeking clinical experience, especially surgical, grew a vicious circle. In order to attract the best men in the quiz the attending surgeons in the leading hospitals gave more and more of their operative work to the relatively inexperienced surgical intern during the last four to six months, when he was called House Surgeon. The quiz men naturally went for the surgical services offering the most operating—a rather characteristic trait of young aspiring surgeons. This resulted in turning out a great many hospital graduates with limited training and immature operative experience who either began a general practice with what little surgery they could get, or did no surgery at all, for there was not a great deal of surgery to be done in those days. Furthermore, the younger attending surgeons, similarly trained, got little further operative experience other than assisting the house surgeons, who expected and got the lion's share of the operations that had not been taken over by the Senior Surgeon.

This all had an unfortunate and pernicious effect in retarding the idea of residency training in general surgery. For the Senior Attending Surgeons felt that it would jeopardize the obtaining of the top quiz men for their internships, the younger Attending Surgeons felt that it would deprive them of what little operative experience they were getting, and, of course, the surgical interns were convinced that their training would be ruined by the advent of the resident system.

That Halsted had the best quiz in New York was due to the fact that he made it much more than a memorizing discipline for drilling his students to meet the hospital examinations and the peculiarities of the examiners in the different hospitals. He and his associates not only gave them intensive training in anatomy and physiology, but made it possible for them to attend courses in pathology and bacteriology in Welch's Laboratory at the Bellevue Hospital. He had his students attend the surgical outpatient department at the Roosevelt and took them to see bedside patients in the hospital as well.

Nevertheless, it must be said that, ironically, Halsted, who initiated the surgical residency program at the Hopkins, was in a way responsible for the delay of its acceptance in New York. After he left New York and began his surgical work in 1889, and later his residency training program, a number of his former associates in New York went to Baltimore to see him work and to inspect his training program. They had known him as a bold and rapid operator. Here in Baltimore they saw him working slowly and deliberately, taking an hour or two to repair a hernia, an operation that he and they had done in twenty minutes. They thought he puttered and wasted his time in details which they did not understand. But what they could not tolerate was his submitting to what they called the tzarlike attitude of his resident, who, they said, assumed the responsibility of the chief and took over most of the operative schedule. This was at the time Cushing was resident, when Halsted was more interested in the thyroid and other problems in the Hunterian Laboratory.

These men came away more prejudiced than ever against the resident system, and this prejudice held until the late nineteen twenties, to the great delay of sound surgical training and original surgical research in New York City. In 1921, when I began the surgical residency training program at the Presbyterian at the time that I was appointed to the Chair of Surgery at Columbia, I did not call the men residents but, to initiate the program with as little prejudice as possible, called them Fellows in Surgery. It was not until George Heuer came to the New York Hospital and began his residency training there, strictly along Halstedian lines, that we called our men residents. Now all the worthwhile surgical services in New York have residents, both because they appreciate the value of the residents to the attending surgeons as well as to the patients, and because they wish to have their men qualify for the American Board of Surgery. The story of the American Board is an interesting one, and will be written some day because of its relation to residency training, conceived and started by Halsted.

During his New York period, he demonstrated his sound surgical judgment and his operative ability in the several hospitals to which he had been appointed, in his teaching at the Physicians and Surgeons, and in his quiz. It is interesting to note that with his increasing duties and responsibilities he found time to think independently and to make original contributions. During the period of 1882 to 1886 he contributed some twenty-one papers to medical societies or medical periodicals. Many of these were in the nature of case reports and operative results, yet some of his studies and observations were of fundamental importance. His reports from the Bellevue Hospital on the use of traction and countertraction in the treatment of fractures; the microscopic study of urethral smears following urethral irrigation with bichloride solution in gonococcal infections, treated at the Roosevelt outpatient department; his original method of arterial reinfusion of the blood of carbon monoxide poisoned patients, after it had been withdrawn and oxygenated, as he used it at the Chambers Street Hospital; and, finally, his papers on the use of cocaine, epoch-making in their effect on the development of the new subject of local anesthesia—these were examples of his original thinking and broad outlook.

Two experiences of this period illustrate his intrepid action in the face of emergency under trying personal circumstances. In 1881, soon after his return to New York, he went to Albany to see his sister who was about to have her first child. On arrival he found that she had been delivered but was pulseless and dying of a post-partum hemorrhage.

His own account follows: "After checking the hemorrhage I transfused my sister with blood drawn into a syringe from one of my veins and injected immediately into one of hers. This was a great risk, but she was so nearly moribund that I ventured it and with prompt result."

This quotation is from a letter written by Dr. Halsted in his last year, 1922, and quoted by Dr. George Heuer who, after careful investigation, considers Dr. Halsted's donor blood transfusion "the first instance in the human being of successful direct blood transfusion by the syringe method."

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A year later Halsted was again faced with an emergency in his own family. His mother became increasingly ill with jaundice and swelling and great tenderness in the region of the gall bladder. She had been under the care of his close friend, Dr. McBride, who had called Sands, Draper, Janeway, and Delafield in consultation.

In the same letter, without commenting on the result of the consultation he wrote: "At 2 A.M. I operated, incised the gall bladder which was distended with pus, and extracted seven stones." His mother recovered from what must have been one of the first successful cholecystostomies for suppurative cholecystitis.

In 1885, we find Dr. Halsted at the height of his New York career, a brilliant surgeon with the experience he had acquired in the best clinics abroad, a fearless, rapid operator in the best hospitals in the city, an indefatigable worker and teacher, a bachelor living with his friend McBride in a house on East 25th Street, where, as genial hosts, they entertained with dinner parties and musicales, an enthusiastic bowler—an expert, when time permitted—a great favorite of such eminent surgeons as Sands, Bull, and Stimson, as well as of his many younger associates.

But within a period of a few weeks in that year 1885 a terrible calamity overtook him. Hearing of the discovery of a new drug which produced anesthesia in the cornea and the conjunctiva, as reported by Koller at the Ophthalmic Congress in Heidelberg, Halsted sent for a large supply of this material. This he did without any knowledge of its side effects. When it arrived he and his associates, Hall and Hartley, began a series of experiments on themselves to determine the anesthetic effect on thir peripheral nerves and on their mucous membranes, especially the nasal mucous membranes when insufflated. Innocently, they tried the drug on some of their quiz men and a few of their friends. While the supply lasted they discovered a number of new effects—with strong and weak solutions, with and without the use of the tourniquet. They and their associates observed that while taking the drug their capacity for prolonged effort was amazingly increased, that food and sleep could be dispensed with and that they seemed to have eliminated fatigue. But with its use came the increased demand for its use, and when the supply was exhausted the inevitable, but then unknown, result of deprivation followed. Halsted and three of his associates, who had innocently become addicts, went all to pieces. Alcohol and other sedatives proved useless. It is a very revealing story that the operative records of Halsted at the Roosevelt and the Presbyterian, as I examined them, stop abruptly.

Dr. William H. Welch, then the Pathologist at the Bellevue Hospital, was a devoted friend and great admirer of Halsted. He quickly realized the seriousness of the problem. The story of what he did for his friend has not as yet been published, probably at his request while he was living, for it must have been a dreadful memory that he did not wish recalled. But now that the great man has gone and all the figures in the drama are historic it should be recorded. Dr. George Woolsey, some two years before he died, told me that Dr. Welch hired a schooner and with three trusted sailors sailed with Dr. Halsted to the Windward Islands and back. This was as great an example of Jonathan-David

devotion as I have ever known. What a tragic experience it must have been for both of them! On their return Dr. Halsted spent a period of months in the Butler Hospital in Providence, and again the next year. The records of that hospital, as I have been able to learn, show that when he left at the end of his second stay there he was entirely free from any evidences of addiction.

Inasmuch as his tragic experience occurred in his New York period, I have had to mention it; because it had such a profound effect on his personality, I should like to comment on it. This sudden break in his intensive activities in New York and his enforced seclusion for months, with the prolonged period of mental anguish and contemplation that it brought him, changed him from an extrovert to an introvert.

Dr. Edward D. Churchill, speaking at the Retirement Dinner given recently to Dr. Evarts Graham in St. Louis, emphasized the significance of Dr. Graham's withdrawal from surgery for a period of three years to study the chemistry that could be applied later to problems in surgical research.

He had this to say: "The motif of Withdrawal and Return is an ancient one. Plato developed it in the famous simile of the Cave. Toynbee dealt with it at length in an analysis of the growth process of individuals and societies. Withdrawal gives opportunity for a man to develop powers within himself which otherwise may remain dormant in a life crowded with the pursuit of limited objectives. No mystical element need be invoked in the withdrawal and return of a scientist, surgeon, and teacher. To such a person withdrawal provides an opportunity for the mind to be receptive because it is released from the pressing demands of the toils and distractions of ordinary life. Exposure of the receptive mind to significant and profound experience makes the prepared mind."

Certainly Dr. Halsted had a receptive mind, and as a result of his innocently acquired addiction and his voluntary hospitalization, he was exposed to a significant and profound experience. It was an experience that was initiated by physical and mental agony, and carried on with superhuman will power before his period of contemplation could begin. But when it began and continued it was productive of his changed surgical ideas and of his new surgical philosophy. It was this macabre combination of fierce struggle and enforced and prolonged contemplation that made such a profound change in his personality.

Had Halsted not gone through this dreadful experience he would have continued as a brillant, overworked surgeon in New York and would undoubtedly have followed the pattern of his day. Instead, this experience resulted in his conceiving and establishing the only original and individual School of Surgery in this country.

Dr. Alfred Blalock, brilliant and worthy successor to the Halstedian Tradition in this, your School and City, aptly epitomized Halsted's translation from New York to Baltimore in a letter to me, in which he said: "I think it is all to Dr. Halsted's credit that he was able to overcome this habit, and it is probably very fortunate for American Surgery that he acquired it."

To me, as a surgeon who in my earlier days saw and did much of the same kind of surgery that was done when Halsted was in New York but who became

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converted later to the Halsted tradition, it has been most interesting to note the trend toward the careful, safe surgery of the Halsted School in recent years, as well as the adoption of the residency training program in the leading surgical clinics of this country. And this has not been a blind following of Halsted's teaching but the appreciation of the basic principles of his surgical philosophy which has resulted in the accomplishments in the surgery of today that were not contemplated by Halsted or his earlier residents.

Some of his most profound philosophy was expressed by him in very cryptic fashion, as in his casual remark: "Silk should not be used that does not break easily."

His whole philosophy of wound repair is summed up in those ten words, but little understood or appreciated by too many surgeons.

In closing, I wish to acknowledge my indebtedness to Dr. MacCallum's biography of Dr. Halsted, to conversations I had with Dr. George E. Brewer and Dr. George Woolsey in their later years, and to Dr. George Heuer for many of the facts recorded in this communication. Finally, I wish to express my great appreciation of the privilege of taking part in the celebration of the Centenary of the birth of the great surgical philosopher, William Stewart Halsted.

# RENAL LESIONS AND FUNCTION FOLLOWING PROLONGED EXPERIMENTAL HYPOTENSION

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ALTHOUGH the occurrence of decreased renal function following shock has been recognized clinically in man.[1-4] and although it is well established that there is marked diminution in renal function during the period of shock or hypotension in man and the dog,[1, 2, 5-8] the demonstration of the presence of pronounced renal dysfunction or renal lesions following recovery from experimental shock or hypotension has remained a difficult problem.[9-13] Dogs either die acutely during the period of shock or, if recovery from shock occurs, promptly regain essentially normal renal function.[9, 10, 12-14]

There has been some disagreement regarding the type of renal lesions which shock produces in man. The widespread use during the past few years of the term "lower nephron nephrosis" has tended to focus attention on the lower nephron as the primary site of degeneration and loss of function in the kidney in acute tubular insufficiency of all types.[15] Mallory and co-workers[16] reported that in human kidneys the earliest microscopic abnormality resulting from shock is vacuolization of the ascending limb of the loop of Henle. Yet Bell,[17, 18] Moon,[19] and Herbut[20] have maintained that the site of renal degenerative changes due to shock in man is in the proximal convoluted tubule. Furthermore, in producing fatal acute renal insufficiency by occlusion, for more than three to four hours, of the renal artery of one kidney after removal of the other in the dog, most investigators have reported that the degenerative changes seen microscopically are most severe in the proximal convoluted tubules.[21-24] The degenerative changes, however, involve all parts of the tubule. Even in pigment nephropathies, degenerative changes are seen in segments of the nephron other than the distal tubule.[25-28] The role of shock in the formation of casts in renal tubules and the significance of tubular obstruction by casts in the production of acute renal insufficiency are additional problems that are not entirely solved as yet.

In order to mobilize compensatory hemodynamic reactions to a marked degree for 6 hours or longer and yet not cause death for several hours thereafter, the mean arterial blood pressure of dogs in this series was maintained at about 70 mm. Hg by controlled hemorrhage. The objectives were: (1) to study the effect of prolonged hypotension per se on the kidney, (2) to study renal function several days after hypotension was terminated by returning to the dog the blood previously removed, and (3) to determine the lesions that might occur in the kidney.

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## METHODS

Dogs weighing 7 to 22 kg. were used in this study. Hypotension was produced by cannulation of a femoral artery of the dog under aseptic precautions and bleeding through a short length of Polythene tubing into a reservoir bottle in which the pressure was held at 70 mm. Hg. The reservoir bottle contained at the start 15 c.c. of 10 per cent solution of sodium citrate, and 30 c.c. of heparin solution (10 mg. per 100 c.c.) in physiologic saline solution was added slowly to the blood as it passed through the tubing when the initial rapid bleeding occurred. A slow drip of heparin in physiologic saline solution (10 to 20 mg. per 100 c.c.) into the tube leading to the reservoir was continued throughout the experiment. Furthermore, in some dogs, systemic heparinization was produced throughout the experiment by a continuous drip of heparin solution (10 mg. to 20 mg. per 100 c.c.) into one of the foreleg veins. No difficulty with clotting occurred when these procedures were carefully followed. The blood was filtered prior to reinfusion. Aseptic precautions and sterile equipment were used. The dog was anesthetized initially with pentobarbital sodium given intravenously. Small quantities of the anesthetic agent were administered when needed during the experiment since the condition of the animals appeared better when this was done to prevent struggling and hyperventilation. Actually, the amount of anesthetic agent required during the period of hypotension was not very great, for the hypotension itself produced stupor. The urinary bladder of the dog was catheterized to determine the volume of urine during each experiment.

In one group of 8 dogs renal function was evaluated prior to, and for several days to several weeks after, periods of hypotension varying in duration from seven to twenty-three hours. The studies of renal function included determination of urinary volume, routine urinalysis, and determination of plasma nonprotein nitrogen, and renal clearances of creatinine and para-amino hippurate (glomerular filtration rate and effective renal plasma flow, respectively). The clearances were determined on the dog on the day preceding, and on the second day following, the termination of hypotension. The determinations were made on trained female dogs in the postabsorptive state after the administration of a priming dose and during continuous intravenous infusion of a solution of creatinine and para-amino hippurate (PAH) under conditions of water diuresis produced by giving by gavage 50 c.c. of water per kilogram of body weight. Plasma levels of 1 to 3 mg. of PAH per 100 c.c. and approximately 20 mg. of creatinine per 100 c.c. were maintained during the clearance. The concentration of creatinine in the urine and plasma was determined by the method of Bonsnes and Taussky,[29] using an acid tungstate protein-free filtrate of plasma. The concentration of PAH in the urine and plasma was determined by the method of Smith and associates,[30] using a protein-free filtrate of plasma prepared with 5 per cent solution of trichloracetic acid. The clearance values reported are average values for 3 to 6 consecutive periods of 10 to 30 minutes each. The clearance data presented are uncorrected for extraction ratios or body surface areas. The kidneys of these dogs were removed 5 to 11 days later for study. In 7 additional dogs renal

function studies other than clearances were carried out and the kidneys were removed for histologic study 5 to 9 days after the period of hypotension had been produced.

Another group of 13 dogs was used for the study of early histologic changes in the kidney. This group included those dogs that died during, or shortly after, the period of hypotension, those in which hypotension was continued until death, and those in which the experiment was intentionally terminated during the period of hypotension. The duration of hypotension in this group varied from 6¼ to 26 hours. The tissues were immediately fixed in 10 per cent solution of formalin and were stained with hematoxylin and eosin for microscopic study.

## RESULTS

*Characteristic Features Occurring During the Period of Hypotension.*— Hematuria, albuminuria, and severe oliguria or anuria were consistently observed during the period of hypotension. The gross hematuria which appeared during the period of hypotension persisted sometimes for 2 to 3 days after the termination of hypotension.

TABLE I. VOLUMES OF URINE DURING AND IMMEDIATELY AFTER THE PERIOD OF HYPOTENSION

| | PERIOD OF HYPOTENSION | | PERIOD IMMEDIATELY AFTER REINFUSION OF BLOOD | |
|---|---|---|---|---|
| DOG | DURATION (IN HR.) | URINE (IN C.C.) | DURATION (IN MIN.) | URINE (IN C.C.) |
| 1 | 7 | 30 | 15 | 50 |
| 2 | 7 | 0 | 15 | 50 |
| 3 | 7 | 38 | 15 | 30 |
| 4 | 20 | 84.5 | 30 | 36 |
| 5 | 21.75 | 0 (first 6 hr.)<br>73 (last 15.75 hr.) | 45 | 40 |
| 6 | 21.5 | 5 | 25 | 0 |
| 7 | 22.25 | 5 (first 6.5 hr.)<br>68 (last 15.75 hr.) | 150 | 40 |
| 8 | 22 | 79 | 110 | 50 |
| 9 | 19 | 170 | 45 | 50 |
| 10 | 19 | 6 (first 7.5 hr.)<br>91 (last 11.5 hr.) | 90 | 120 |
| 11 | 18.25 | 78 | 60 | 5 |
| 12 | 18.5 | 18.5 (first 9.5 hr.)<br>35 (last 9 hr.) | 60 | 30 |

The flow of urine ceased as soon as the blood pressure approached 70 mm. Hg with the initial bleeding. A very slow flow of urine usually appeared after several hours, but marked oliguria persisted throughout the period of hypotension (Table I). In the more prolonged (24 hour) period of hypotension, the flow of urine slightly increased during the last 5 to 10 hours. The volume of urine produced during the experiment served as a relatively reliable index of the extent of renal damage accompanying the hypotension. The dogs with kidneys showing the most severe damage excreted less than 30 c.c. of urine during a period of hypotension lasting approximately 24 hours. Dogs with minimal evidence of damage excreted nearly 200 c.c. of urine during the 24 hours. The specific gravity of urine formed during the period of hypotension

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was usually high (1.035 to 1.040). However, in a few instances in which renal damage was later found to be extensive, the specific gravity was low, that is, 1.010 to 1.020.

The rapid increase in the volume of urine immediately following reinfusion of blood and the concomitant increase in blood pressure convincingly demonstrated the importance of the level of the arterial blood pressure in the formation of urine. A total varying from 0 to 38 c.c. of urine was produced during periods of hypotension lasting 7 hours, but an equal or greater volume of urine would be formed during the first 15 to 30 minutes following reinfusion of blood (Table I). However, dogs whose kidneys sustained severe damage during the longer periods of hypotension often excreted only a few cubic centimeters of urine during the first 15 to 30 minutes following the reinfusion of blood.

In general, as the mean arterial blood pressure was lowered initially to 70 mm. Hg, a volume of blood of about 25 to 45 c.c. per kilogram of body weight rapidly flowed from the artery of the animal into the reservoir bottle. During subsequent hours, the volume of blood in the reservoir gradually increased until about 50 to 60 c.c. per kilogram of body weight was removed. Thereafter the volume in the reservoir either remained constant or oscillated within ±100 to 200 c.c. Later, toward the end, in some of the longer experiments, the dog took back from the reservoir several hundred cubic centimeters of blood. Dogs usually hyperventilated during a good portion of the period of hypotension and were very pale.

Nine dogs either died during the period of hypotension or the hypotension was intentionally continued until death occurred. In 4 of the 9 dogs the death accompanied the abrupt cessation of respiration or irregular rapid heart action or both. No blood was taken back by any of these 4 dogs before death, which occurred at a mean arterial blood pressure of 70 mm. Hg. In these dogs, irreversible shock, as defined by Wiggers,[31] did not occur.

In 5 of the 9 dogs evidence of irreversible shock did occur. After the volume of blood in the reservoir had remained constant for several hours, the dog rapidly took back all the blood from the reservoir and death occurred. If blood in the reservoir was rapidly reinfused after the animal was seen to be taking it back rapidly, death occasionally was avoided, but in most cases death occurred, nevertheless. After the dog had taken back all of its blood, administration of additional blood and fluids failed to maintain life. In 1 dog weighing 11.5 kg., 400 c.c. of blood and 800 c.c. of physiologic saline solution in addition to the return of all of the blood in the reservoir failed to maintain life. Eventually, at this stage, it becomes impossible to infuse fluids rapidly enough to maintain life.

Two dogs died within 5½ hours after return of the blood from the reservoir. If dogs survived the period of hypotension and the first few hours thereafter, complete recovery usually occurred.

Pulmonary edema was a rare occurrence in these experiments.

*Renal Function Following a Prolonged Period of Hypotension.*—Evidence of renal dysfunction a significant degree was not observed after hypotension of seven hours' duration. Albuminuria and hematuria occasionally continued

for a day or two, but the volumes of urine were not significantly altered and plasma levels of nonprotein nitrogen did not exceed 48.6 mg. per 100 c.c. The values for creatinine clearance did not change significantly when determined 48 hours after the termination of hypotension, but a decrease in PAH clearances occurred in several dogs (Table II).

TABLE II. EFFECT OF PROLONGED HYPOTENSION ON VALUES FOR RENAL CLEARANCE AND PLASMA VALUES FOR NONPROTEIN NITROGEN

| DOG | | HYPOTENSION (IN HR.) | EFFECTIVE RENAL FLOW (IN C.C./MIN.) | | | | GLOMERULAR FILTRATION, C.C./MIN. | | PLASMA NONPROTEIN NITROGEN, MG./100 C.C. | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | PLASMA | | BLOOD | | | | | |
| NO. | WT. (IN KG.) | | BEFORE* | AFTER* | BEFORE* | AFTER* | BEFORE* | AFTER* | BEFORE* | AFTER* |
| 1 | 22 | 7 | 309.6 | 237.3 | 491.4 | 379.7 | 82.5 | 75.1 | 29.6 | 36.0 |
| 2 | 14 | 7 | 225.6 | 120.6 | 398.6 | 260.5 | 54.4 | 54.6 | 25.0 | 26.6 |
| 3 | 15 | 7 | 108.2 | 125.0 | 224.9 | 230.6 | 52.9 | 44.9 | 42.8 | 41.0 |
| 4 | 12 | 20 | 245.7 | 137.7 | 478.0 | 272.6 | 77.3 | 48.7 | 35.0 | 53.2 |
| 6 | 12 | 21.5 | 165.3 | 66.6 | 325.4 | 114.6 | 59.5 | 17.1 | 35.2 | 36.4 |
| 7 | 9 | 22.25 | 134.5 | 15.8 | 241.9 | 34.9 | 38.9 | 9.6 | 27.6 | 56.1† |
| 8 | 8.7 | 22 | 184.3 | 53.7 | 310.8 | 87.3 | 60.2 | 35.1 | 26.5 | 27.6 |
| 13 | 18 | 23 | 166.3 | 113.0 | 327.4 | 189.6 | 57.7 | 42.2 | 34.0 | 110.0‡ |

*Determinations of renal clearance and plasma nonprotein nitrogen were made 24 hours before and 48 hours after the period of hypotension.

†Nonprotein nitrogen increased to 110 on the fifth day after the period of hypotension, when the dog appeared very ill and the kidneys showed bilateral cortical necrosis.

‡Nonprotein nitrogen decreased to 40 two days later.

After recovery from hypotension of longer duration (up to 23 hours), there was definite evidence of renal dysfunction, although frequently the reduction in renal function was not as great as might be anticipated. Hematuria and albuminuria usually persisted for several days. The volume of urine excreted varied, but, in general, remained surprisingly high. In nearly one-half of the dogs, little change in the volume of urine occurred; in some there was definite polyuria, and in others oliguria resulted. Oliguria was usually found in those cases in which degenerative changes occurred in the entire renal cortex, while those dogs whose kidneys contained scattered areas of degenerative lesions with large amounts of intervening normal tissue either had polyuria or no change in volume of urine. Increases of plasma nonprotein nitrogen usually were small but values up to 110 mg. per 100 c.c. were encountered. The specific gravity of the urine varied considerably as a result, at least in part, of the varied volumes of urine. The specific gravity was generally low in cases in which there was polyuria, and within normal range in other instances. In some dogs having oliguria, values for specific gravity in the vicinity of 1.020 were noted several days after the period of hypotension.

Values for renal clearance determined 48 hours after hypotension of 20 to 23 hours' duration were often markedly reduced and sometimes in a range comparable with those seen in man in acute renal insufficiency (Table II).[32-35] Clearance values usually approached normal, however, in the 4 dogs in which the determinations were made 1 to 3 weeks after the period of hypotension.

Especially noteworthy was the inhibition of diuresis which occurred after the period of hypotension in 3 of 5 dogs. Renal clearances were determined before and after hypotension of 20 to 23 hours' duration. The 50 c.c. of water per kilogram of body weight given to produce diuresis for the study of clearances failed to cause the usual diuresis and the administered fluids were only slowly excreted. The average volume of urine for each 10-minute clearance period in 1 dog averaged 89.2 c.c. before the period of hypotension whereas, after such period and when exactly the same procedure was used, the average 10-minute volume of urine was only 13.9 c.c. In another dog the average 10-minute volume of urine before the period of hypotension was 45.2 c.c., while after the period of hypotension and after treatment of the dog in exactly the same manner the greatest volume of urine that could be obtained during a 30 minute period was 14.5 c.c. In the third dog, the average 10 minute volume of urine before the period of hypotension was 40.4 c.c., but the greatest volume during 30 minutes after the period of hypotension was 10.2 c.c. The specific gravity of the urine was approximately 1.020. A similar failure of diuresis after administration of water has been reported to occur following the resection of approximately 75 per cent of the renal tissue of dogs.[36]

In general, the dogs survived if they lived through the period of hypotension and the first few hours thereafter. However, in 1 dog the experiment was terminated on the fifth day after the period of hypotension because its condition was poor and the plasma value for nonprotein nitrogen was 110 mg. per 100 c.c. the cortices of the kidneys of this dog showed severe necrosis, and severe degeneration was also present in some areas of the medullas of these kidneys (Fig. 1).

The values for hemoglobin in the blood and hematocrit readings were approximately the same preceding, and 48 hours after, the period of hypotension. In no instance was there evidence of hemoglobinemia.

*Pathologic Changes.*—The renal lesions varied tremendously in regard to the amount of tissue involved, the distribution of the lesions, and the severity of the lesions. In general, all segments of the tubules were affected but the proximal convoluted tubules usually showed the severest degenerative changes. However, the over-all pathologic picture was that of generalized tubular degeneration in the cortex, and to emphasize the localization of degeneration to any one tubular segment would be inconsequential. Variations ranging from small focal areas of mild degenerative tubular changes to bilateral cortical necrosis were found. The most common pattern of lesions found was that of numerous cortical infarcts in the kidney (Fig. 2). The severity of degenerative changes within these infarcts also varied greatly. Microscopic evidence of some damage was found in every kidney.

In 1 dog, marked renal lesions were found after 6¼ hours of hypotension. This was the earliest time following the onset of hypotension that pronounced renal lesions were found. However, this was unusual and degenerative changes usually were early and mild in an animal dying at this

time. Otherwise, it was not possible to relate closely the severity of the lesion to the duration of hypotension, nor to whether death or recovery occurred from hypotension. It was not possible to correlate the lesions with the type of

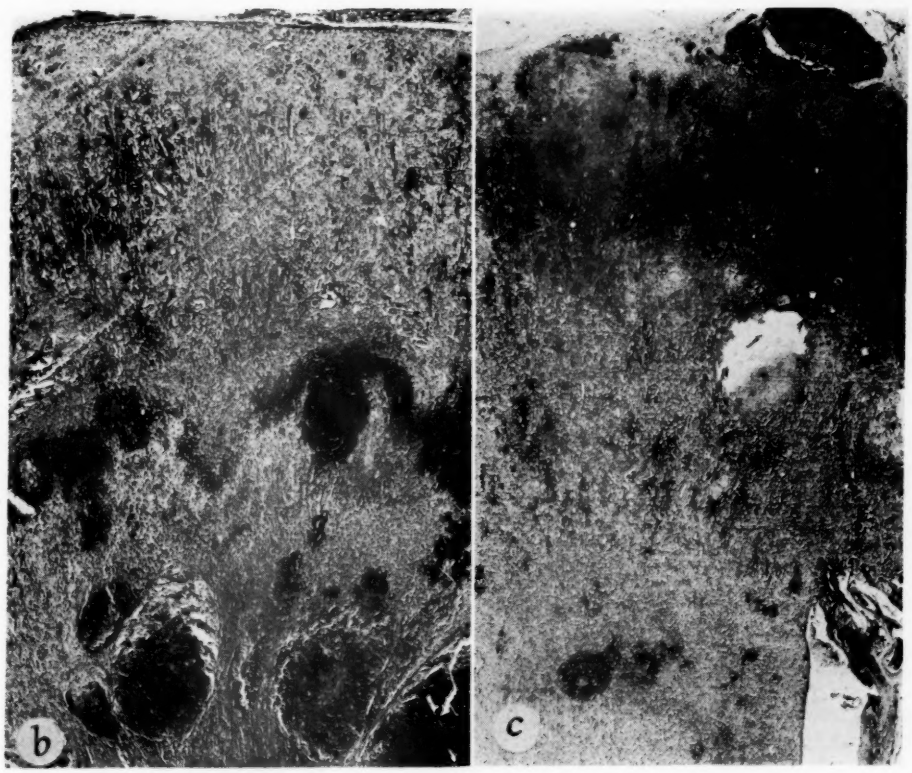


Fig. 1.—*a*, Severe necrosis in a kidney removed 5 days after termination of a 22-hour period of hypotension. *b*, Section from an area in the kidney in *a*, showing coagulation necrosis (infarction) of the cortex and medulla but only degeneration without infarction in the corticomedullary region (hematoxylin and eosin, ×8½). *c*, Section from the contralateral kidney showing infarction of the cortex but a relatively uninvolved medulla (hematoxylin and eosin, ×7).

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death that occurred during the period of hypotension, be it from cardiorespiratory failure or irreversible shock. The extent of the lesions was unpredictable other than for the fact that, in general, more than 7 hours of hypotension were required to produce detectable lesions when the kidney was studied immediately after the period of hypotension or several days later. However, if the volume of urine during the period of hypotension was very low, extensive renal lesions could be anticipated.

Fig. 2.—Numerous ischemic areas in the cortex with a large hemorrhagic infarct in the kidneys of a dog dying from irreversible shock after 12 hours of hypotension.

*Changes in Kidneys Studied Immediately After a Prolonged Period of Hypotension.*—Grossly the kidneys usually appeared mottled, pale areas of varying size being present on the surface. When the cross section of such kidneys was inspected, wedge-shaped or rectangle-shaped white areas were seen in the cortex (Fig. 2). These varied in number and size. Sometimes they were restricted to cortical tissue superficial to the juxtamedullary region, other times they involved all of the cortex and juxtamedullary region, and infrequently the upper part of the medulla also appeared involved along with the corresponding cortical tissue superficial to it. In some cases the entire surface of the kidney was uniformly pale, and on cross section the cortex was pale and granular in contrast to the congested dark red or purple medulla (Fig. 3). Some kidneys were nearly normal on gross inspection although their cortices were slightly pale and their cut surface had a granular appearance. Microscopically, the cortices of these latter kidneys often presented granular degeneration and karyolysis of nuclei of the cells of the cortical tubules, although the glomeruli and medulla were apparently normal.

As previously mentioned, the extent of involvement of the kidneys as well as the microscopic features of the lesions varied greatly. The degenerative changes were usually limited to the cortex. The lesions sometimes involved the entire cortex, sometimes only certain areas of it. When the degeneration was most severe and involved the entire cortex of each kidney, the picture of bilateral cortical necrosis was seen. When only segments of the cortex were involved the lesions appeared as cortical infarcts, which varied from a few to many in number. The severity of degeneration varied from place to place in

the same kidney. It is to be emphasized that most frequently there were relatively normal areas of renal tissue between areas involved by degenerative changes. The microscopic changes within an area of involvement sometimes consisted of mild hydropic degeneration of the tubular cells (Fig. 4). Frequently, severe degeneration of tubules in the involved area had occurred, but

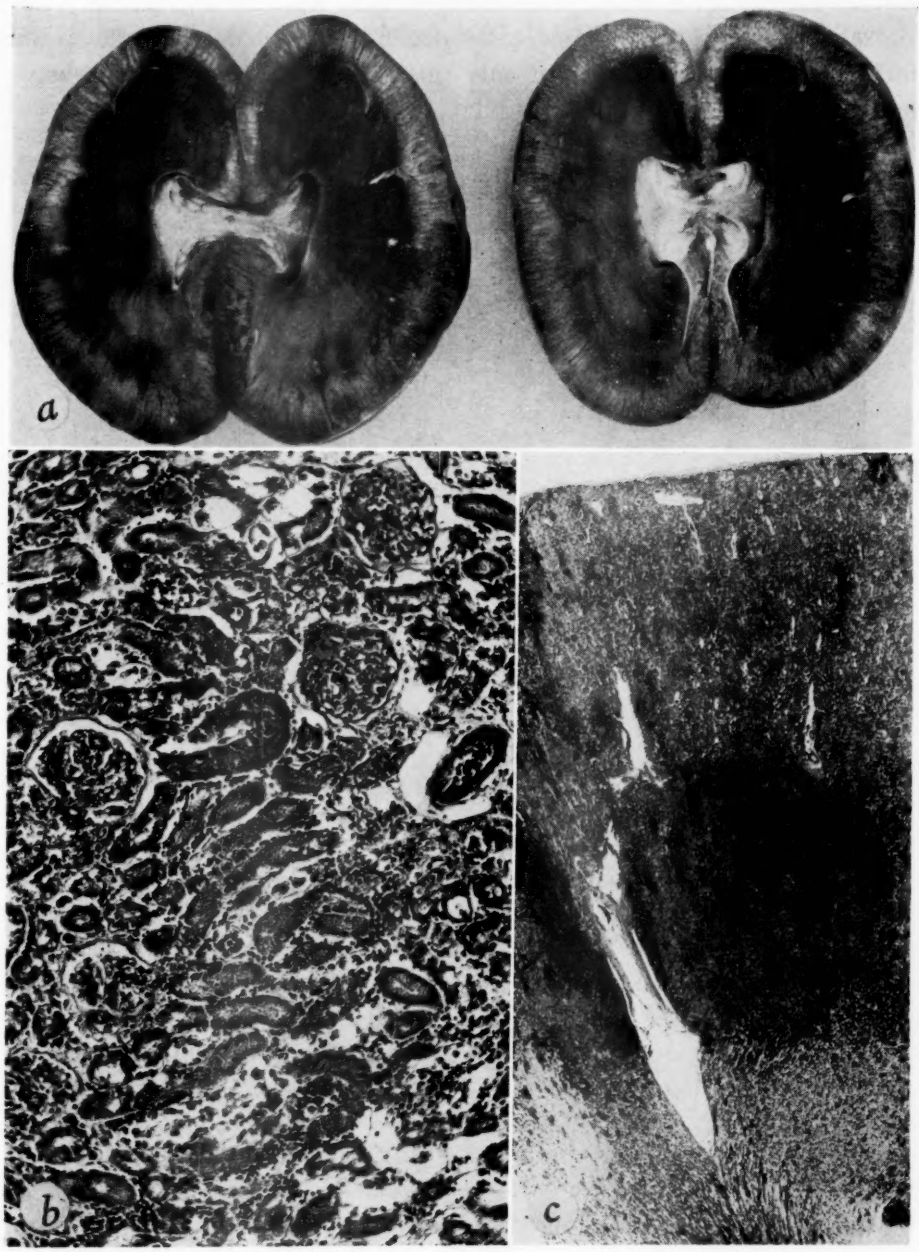


Fig. 3.—Kidneys of a dog which died in irreversible shock after 22¼ hours of hypotension. *a*, Ischemic cortices and congested medullas. The medullas were dark purple while the cortices were pale. *b*, Section showing severe necrosis of all tubules, minimal changes in the glomeruli, and interstitial edema in cortex (hematoxylin and eosin, ×100). *c*. Section showing that congestion is especially marked in the corticomedullary region (hematoxylin and eosin, ×6).

the glomeruli either were apparently normal or had undergone relatively mild changes. The abnormality in the glomeruli frequently was that of endothelial swelling and reduction in caliber of the capillaries. In a number of instances, severe degeneration was present in all tubules as well as in many glomeruli of the involved cortex. Even though the degeneration of the tubules of the cortex was severe, the medulla below such areas usually appeared essentially normal histologically (Fig. 5). In a few cases degenerative changes in the proximal convoluted tubules were present in only small scattered foci. Hemorrhagic infarction occasionally occurred.

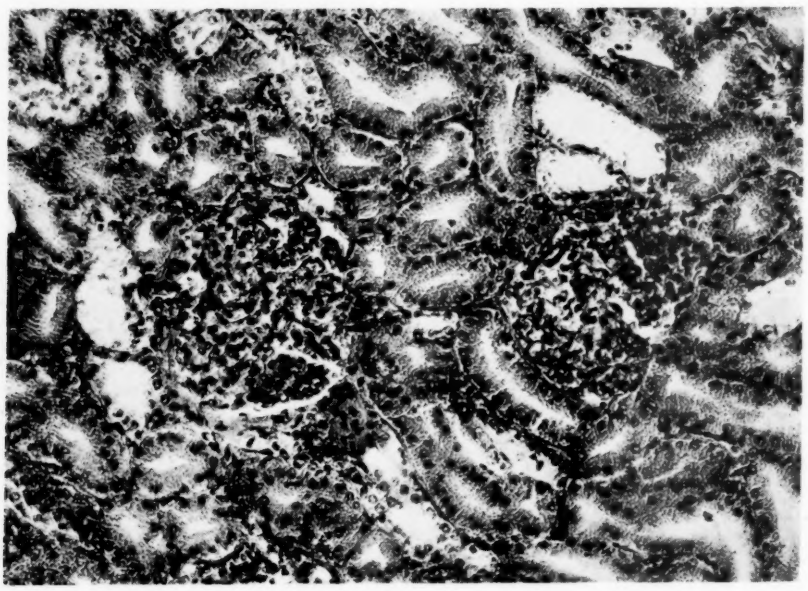

Fig. 4.—Section showing early or minimal degenerative changes in the tubules of the renal cortex of a dog after 16¼ hours of hypotension. All segments of the nephron are involved. Marked endothelial proliferation and narrowing of the lumens of capillaries in the glomeruli are present (hematoxylin and eosin, ×180).

In a few kidneys removed from dogs which died after a period of 7 hours or more of hypotension, hydropic and granular degenerative changes were seen in the ascending limb of the loop of Henle, the distal convoluted tubules, and collecting tubules, occasionally also associated with pyknotic nuclei. The cells of the tubules sometimes appeared to be separated from their basement membrane. However, mild hydropic changes in the cytoplasm of cells of the proximal convoluted tubules were usually present concomitantly. These changes were similar to those found 1 to 4 hours after death in a study of post-mortem changes in dogs' kidneys.[37] Similar lesions have also been found in kidneys removed immediately after 4 hours of complete occlusion of the renal artery. These changes may be similar to those described by Mallory and co-workers[16] as being the earliest changes in the kidney due to shock. Of course, cells of the ascending limb of the loop of Henle sometimes appear vacuolated normally, but the changes under discussion were more pronounced and appeared to represent true degeneration. However, after more prolonged

hypotension or in kidneys studied 1 day or more after occlusion of the renal artery for 4 hours, the severest degeneration was then in the proximal convoluted tubules. In these latter kidneys, the severe hyaline degeneration in the epithelium of the proximal convoluted tubules was often present with complete or nearly complete loss of nuclear staining while the distal tubules showed only hydropic degeneration with nuclei essentially intact. Thus, although all segments of the tubules showed degenerative changes and although

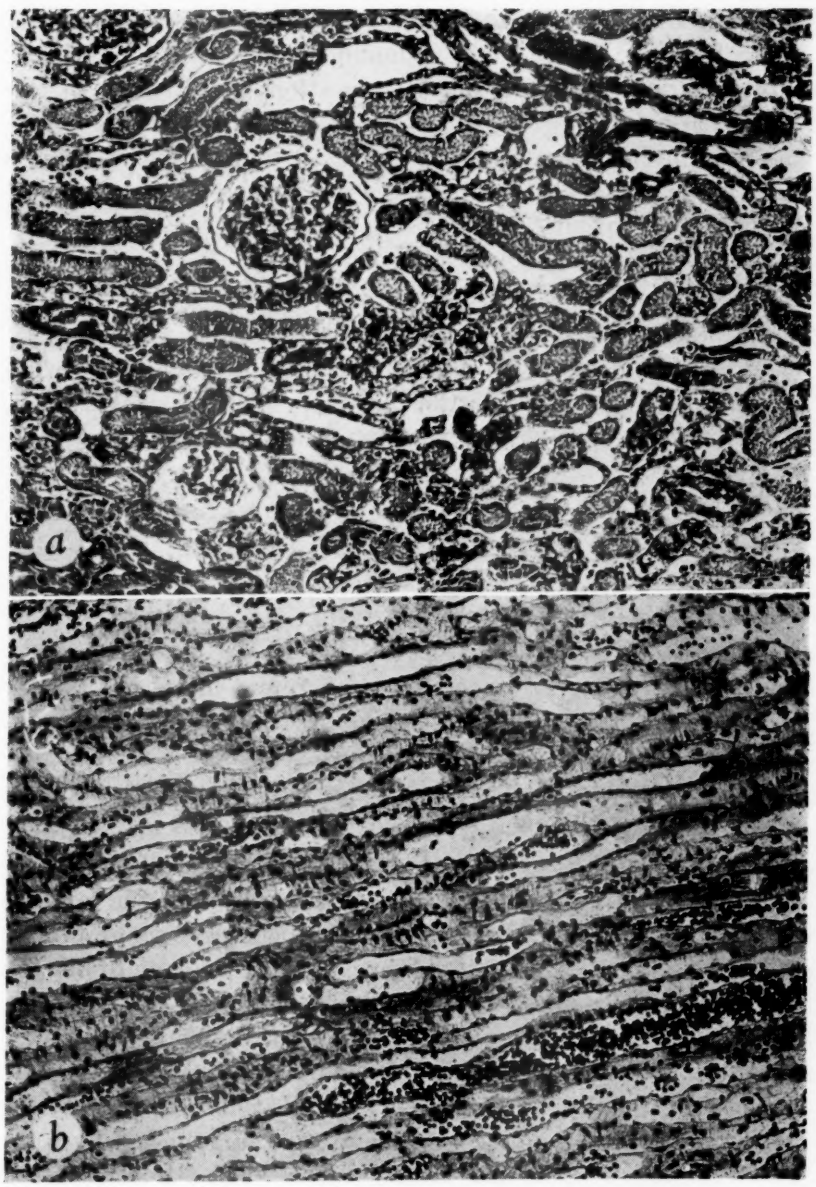


Fig. 5.—*a*, Section showing necrosis of the tubules of the kidney of a dog dying in irreversible shock after 11½ hours of hypotension (hematoxylin and eosin, ×125). *b*, Section from medulla directly below the cortex shown in *a*, demonstrating an essentially normal histologic appearance of the medulla, except for congestion and interstitial edema (hematoxylin and eosin, ×125).

microscopically degeneration may not have been quite so evident in the proximal tubules early in the course, the most complete degeneration following ischemia was seen microscopically in the proximal convoluted tubules.

Degenerative changes were usually absent or mild in the medulla beneath the juxtamedullary region. Occasionally, however, the medulla showed severe degenerative changes below large areas of the cortex which were severely damaged (Fig. 1). The medulla usually was markedly congested. This congestion frequently involved all of the medulla although at times only certain segments of it were congested. Casts were often present in the medulla. The location of the most pronounced vascular congestion was the juxtamedullary region, and sometimes congestion was limited to this region (Fig. 3). One striking observation was that the juxtamedullary region occasionally was normal or at least showed the least damage whereas the adjacent cortex was necrotic (Fig. 1). In fact, in several instances the medulla below and cortex above this juxtamedullary region were completely necrotic, yet the glomeruli were intact and degenerative changes were only moderate in the tubules in the juxtamedullary region.

In a few kidneys the degenerative lesions consisted for the most part of focal areas of degeneration of proximal convoluted tubules deep in the cortex between medullary rays and not in the periphery of the cortex. This localization of lesions to the deeper part of the cortex was probably fortuitous, for in other cases similar focal areas of damage were present in the more peripheral part of the cortex.

The variations in the distribution, severity, extent, and type of degenerative changes were probably the result of variations in the segments of the renal vascular bed involved and the severity and duration of the ischemia of the different areas of renal tissue.

No evidence of bacterial infection was found in these kidneys. The kidneys of 2 dogs which were anesthetized with phenobarbital sodium for 1 to 3 days after laparotomy failed to show evidence of histologic damage. This suggests that prolonged anesthesia per se is not a significant factor in the production of the renal lesions.

*Changes in Kidneys Removed 5 to 11 Days After a Period of Prolonged Hypotension.*—Kidneys removed 5 to 11 days after a period of hypotension showed the same variety of gross and microscopic lesions as described in foregoing paragraphs. In addition there were varying degrees of fibrosis, an infiltration of plasma cells and lymphocytes in the lesions, and scattered areas of calcification. Mitotic figures were frequently seen in tubules of extensively damaged kidneys. Grossly, the kidneys removed several days after a period of hypotension occasionally were swollen. At this time a degenerated proximal tubule often was a mass of pink hyaline material surrounded by a ring of flat cells (Fig. 6). A striking feature was the presence of this hyaline material, sometimes with degenerate nuclei still in it, in the lumen of the distal and collecting tubules, the epithelium of which occasionally showed degenerative changes. These casts, together with casts of a light pink-staining material,

frequently gave a picture resembling that described as characteristic of a phase of the so-called lower nephron nephrosis. However, this histologic picture seen in the "hypotension kidneys" was not uniformly present throughout the kidney. (The term "hypotension kidney" is used in this paper to refer to kidneys subjected to a prolonged period of hypotension.) The damage was not limited to the lower nephron but actually was most severe in more proximal tubular segments. Apparently, hyaline casts in the distal nephron and collecting tubules originated from cells of more proximal portions of the tubule which had undergone hyaline degeneration and which had, in part, passed down the tubule.

Fig. 6.—Section showing necrosis of the tubules from an area in the renal cortex of a kidney removed 5 days after termination of a 19 hour period of hypotension. It may be noted that degeneration appears more severe in the proximal than in the distal convoluted tubules, but the glomeruli appear relatively normal. An infiltration of inflammatory cells has occurred (hematoxylin and eosin, ×135).

*Distribution of India Ink in the Kidneys After a Prolonged Period of Hypotension.*—In 2 dogs during the period of hypotension and in another 2 dogs 5 days after the termination of hypotension, India ink was injected through a cannula passed retrograde via the femoral artery into the aorta. No ink could be found in areas of the cortex in which coagulative necrosis or severe degeneration which involved the glomeruli had occurred (Fig. 7). Ink did usually appear in vessels and glomeruli in areas where glomeruli remained intact although severe degenerative lesions of tubules in the same areas were present.

A generalized deviation of the distribution of ink away from the cortex with its appearance only in the juxtamedullary glomeruli and medulla, as described by Trueta and associates,[38] was not observed in hypotension kidneys. In fact, the opposite was sometimes seen, that is, the presence of ink in the cortex and not in the medulla when ink was injected during the period of

hypotension which had been maintained for 6 hours. The medulla was sometimes congested and yet no ink appeared in it. Ink did not appear in the cortical infarcts in which complete degeneration had occurred, although ink usually perfused the medulla below such areas (Fig. 7). This latter distribution of ink was observed both when ink was injected during the period of hypotension and when it was injected as long as 9 days after the termination of hypotension. This was not considered to indicate that the cause of the lesions was a deviation in blood flow through the kidney, but this distribution of ink and the degenerative changes in the cortex were the result of permanent occlusion of cortical vessels. This picture was found in both denervated and intact kidneys.

Fig. 7.—Portion of a kidney from a dog in which India ink was injected intravascularly 9 days after hypotension had been maintained for 19 hours. The ink failed to appear in several areas of the cortex (infarcts) even though it was present throughout the medulla beneath the pale areas.

The injections of ink demonstrated that (1) sometimes during the period of hypotension blood flow was restricted to the cortex, yet degenerative changes appeared in the cortex, (2) some areas of degeneration of renal tissue resulted from permanent occlusion of the artery supplying the part, (3) tubular degeneration often occurred even though the artery supplying the tissue was not permanently occluded, (4) frequently there was no blood flowing through congested vessels in the medulla, and (5) the cortical lesions were not primarily the result of a deviation of blood flow through the medulla and away from the cortex.

*Heparinization of the Animal Throughout the Period of Hypotension.*—In 8 experiments the dogs were heparinized by means of a continuous intravenous drip of heparin solution (10 to 20 mg. per 100 c.c.) started before the onset of

hypotension and maintained throughout the period of hypotension. This was given in addition to the heparin drip into the cannula in the femoral artery. Renal lesions similar to those previously described occurred in spite of heparinization.

*Renal Lesions Produced by Hypotension After Renal Denervation.*—The left kidney of 2 dogs was denervated by dissecting away all tissue around the kidney leaving only the artery, vein and ureter. The entire length of the vessels was well stripped of nerves. Two weeks later hypotension was induced for 18½ hours, and 5 days later the kidneys were removed. Identical renal lesions as previously described were found in both the denervated and the intact kidneys.

*Comparison of Renal Lesions Produced by Hypotension With Those Produced by Temporary Occlusion of the Renal Artery and Those Produced by Large Doses of Epinephrine.*—In 24 dogs one or both renal arteries were occluded under aseptic precautions by rubber-shod clamps for 3 to 6 hours. In 4 dogs the kidneys were studied immediately after release of the clamp and in the other dogs the kidneys were removed 1 to 17 days later. The same procedure was used in 23 rats, except that the duration of occlusion of the renal artery was 1 to 3 hours only. Microscopic study of kidneys removed immediately after the release of the clamp disclosed only postmortem types of change. Those studied 24 hours or more after release of the clamp had lesions of identically the same variety and extent as those associated with hypotension (Fig. 8). Occlusion of the renal artery for less than 2 or 3 hours usually produced only degenerative tubular changes while occlusion of longer duration produced coagulation necrosis in the form of infarction or bilateral cortical necrosis. Other workers also have been impressed with the variety of degenerative lesions following temporary occlusion of the renal artery.[39, 40]

India ink injected intravascularly in the manner already described also gave the same patterns of distribution as described for hypotension kidneys. Similarly, ink did not appear in areas of total destruction of tissue, but did appear in areas where degeneration was limited to tubules. The degeneration sometimes involved only the cortex superficial to the juxtamedullary region, or the cortex and juxtamedullary region, and at other times it involved all of the cortex and the upper part of the medulla. However, ink appeared in all areas where degeneration was limited to tubules, when it was injected 24 hours or more after removal of the clamps occluding the renal arteries. In a dog whose left renal artery was occluded for 3 hours 10 days previously, ink appeared for the most part only in the juxtamedullary region with a little ink scattered in the medulla (Fig. 9). Microscopically this kidney showed severe degenerative changes involving the entire cortex, especially peripheral to the juxtamedullary region. This latter pattern of the distribution of ink and the degenerative changes are considered to be the result of permanent occlusion of many of the peripheral cortical vessels, and this pattern of blood flow through the kidney is not considered to be primarily the cause of such lesions.

The degenerative lesions in kidneys in which the artery was temporarily occluded were usually limited to the cortex. This was true even though blood flow returned to the cortex of the kidney shortly after removing the occluding clamp, as evidenced by gross observations and the intravascular injection of India ink. This suggests that the medulla is less susceptible to ischemia than is the cortex. Of course, if the renal artery is permanently occluded, necrosis of the entire kidney, including the medulla, does occur.

Fig. 8.

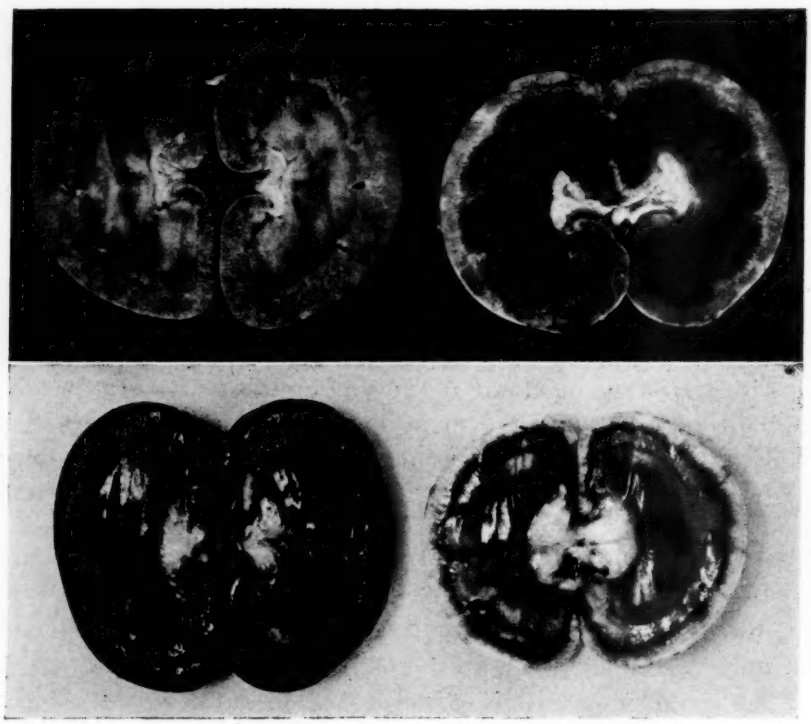

Fig. 9.

Fig. 8.—The artery to the kidney shown on the right was occluded for 3 hours, and 4 days later both kidneys were removed. There is marked congestion of the medulla of the kidney whose artery was temporarily occluded.

Fig. 9.—The artery to the kidney shown on the right was occluded for 3 hours, and then both kidneys were removed 10 days later, after an intravascular injection of India ink. In the kidney to which the artery was temporarily occluded, the ink appears, for the most part, only in the corticomedullary region, while in the control kidney (shown on the left) the ink is evenly distributed.

Urinary volumes following 3 to 4 hours' occlusion of both renal arteries of the dog varied, but often polyuria or mild oliguria occurred, similar to that observed in dogs after a period of hypotension. Occlusion of both renal arteries for 4 to 6 hours in 6 dogs caused death within several days, the dogs having been anuric or severely oliguric until death. The specific gravity of the urine in the oliguric dogs was often in the neighborhood of 1.010.

Dr. Hiram Essex has been studying the effects of large doses of epinephrine in his laboratory in the Institute of Experimental Medicine of the Mayo Foundation. In 8 experiments total doses of 0.8 to 5.54 mg. of epinephrine

per kilogram of body weight were given to dogs as a continuous intravenous infusion of a 1:10,000 solution in 6 dogs, and in 2 dogs as an infusion of increasing concentrations ranging from 1:100,000 to 1:5,000 during the experiment. Death occurred after 3 to 9½ hours. Grossly, kidneys from these dogs showed the same variety and extent of lesions as seen in hypotension kidneys. Microscopically, early degenerative lesions having the same features as those in hypotension kidneys were seen in kidneys of 6 of the dogs. Similar changes were observed in 2 cats and 1 rabbit in similar experiments. The mean blood pressure of the animals usually did not fall below control values during the administration of epinephrine until 20 to 30 minutes before death.

*Lesions in Other Organs Following a Prolonged Period of Hypotension.*—Central necrosis and congestion of the lobules of the liver were consistent findings. This has been frequently observed by others in studying shock and also in clinical cases of acute renal insufficiency, including the crush syndrome and blackwater fever.[16, 41-47] McMichael[43] found that obstruction of the portal vein to a lobe causes central degeneration in lobules of the cat's liver. Widespread vacuolar degeneration was seen in the liver of some dogs studied 5 days after the period of hypotension. In the spleen the disappearance of malpighian corpuscles and the presence of infarcts were often observed. Central necrosis of the liver and splenic infarcts have been found occasionally in man in association with bilateral cortical necrosis of the kidneys.[48] Rarely, small areas of focal necrosis were found in the heart of dogs after a period of hypotension, similar to lesions described by Melcher and Walcott.[49]

## COMMENT

Evidently the renal lesions which occur in prolonged hypotension vary greatly in severity and extent, but are ischemic in origin. The lesions may consist of tubular degeneration only, involving all segments of the tubules but microscopically most severe in the proximal convoluted tubules, if the segment showing the most damage must be named. Such lesions are apparently the result of a prolonged marked reduction in blood flow or a temporary occlusion of the artery supplying the area. However, all tissues in the involved cortex may be completely destroyed. These latter lesions evidently follow permanent occlusion of the artery supplying the tissue, as evidenced by the absence of ink in the lesions after intravascular injections of India ink. The lesions of these varying degrees of severity may involve only segments of the cortex or the entire cortex. When the entire cortex of both kidneys undergoes coagulation necrosis, the condition of bilateral cortical necrosis is seen. Congestion of the medulla is frequently observed but degeneration in the medulla is not common.

It is difficult to be sure of the reason for the occlusion of the vessels. However, we believe that the flow of blood to parts of the kidney which later show degeneration almost completely stops for a number of hours, probably as a result of vasoconstriction and the diminished inflow and pressure of blood going to the kidney following the severe rapid bleeding. In some places this vascular occlusion becomes permanent, as shown by the absence of ink after injection and the development of coagulation necrosis in the form of cortical infarcts or

bilateral cortical necrosis. In other places the great diminution of blood flow persists long enough to produce tubular degenerative changes of varying degrees of severity but blood flow through the vascular bed is restored and the glomeruli are injured little or not at all. Thus, the severity of the degenerative lesions depends on the duration of severe ischemia, and the extent of involvement depends on in which vessels and in how many renal vessels blood flow is severely restricted or abolished.

It is accepted that the renal vessels tend to constrict markedly in a variety of conditions, especially in shock (although renal vascular resistance apparently does not increase significantly in hypotension alone).[1, 2, 5, 7, 12, 31, 50] In shock, effective renal blood flow may be severely depressed even though the systemic blood pressure decreases very little.[51] According to Little and co-workers[50] humoral factors are probably not the cause of this renal vasoconstriction. Renal blood flow determined on the basis of clearances and by direct methods is markedly reduced during periods of hypotension and even more so in shock.[1, 2, 5-7, 31, 52] The fact that the renal lesions found after temporary occlusion of the renal artery are identical with those seen after a prolonged period of hypotension further supports the thesis that the lesions in the latter condition are due to ischemia. Another factor to be considered is the distinctive vascular bed of the kidney, that is, the arterial vessels rapidly end in glomerular capillaries which in turn are followed by still another capillary bed.

We do not believe that the lesions found in our study are due to emboli of extrarenal origin because of the following considerations:

1. Emboli from the cannula or reservoir blood before reaching the kidney would have to pass through at least two capillary beds, namely, the vascular beds of the lower extremity and the lungs.

2. The lesions occurred even though blood removed from the dog to lower the blood pressure to 70 mm. Hg was never returned to the dog. Thus, we do not believe that clots in the blood in the reservoir were the cause of the lesions.

3. Identical renal lesions occur after temporary complete occlusion of the renal artery or after large doses of epinephrine. Others have produced similar renal lesions with vasoconstrictor agents, including epinephrine and vasopressin (pitressin), and consider the lesions to be the result of renal vasoconstriction.[53, 54]

4. The lesions occurred consistently if the hypotension was prolonged beyond approximately 6 hours but rarely occurred when it lasted less than 6 hours.

5. Complete heparinization of the dog and of the blood in the cannula and reservoir did not prevent the occurrence of the lesions.

6. India ink often perfused the glomeruli and vascular bed of kidneys in which severe tubular degenerative changes were found. Evidently the vascular occlusion necessary to produce such lesions was temporary, if complete, or else was incomplete.

We do not believe that these lesions are the result of an intrarenal deviation of blood flow produced by nerve impulses, such as Trueta and associates

postulate.[38] If ink did appear in the medulla and not in the cortex, it was considered to be the result of permanent occlusion of cortical vessels and not primarily a manifestation of deviation of the distribution of blood flow. The renal nerves are not necessary for the production of such lesions since denervated kidneys showed such lesions and others have shown that renal vasoconstriction occurs in shock and hypotension in the denervated kidney.[5] Actually, India ink was found sometimes in the cortex, including the juxtamedullary region, but was absent in the medulla during the period of hypotension. Other studies have convinced us that the restriction of blood flow to the renal cortex, including the juxtamedullary region, sometimes occurs under the following conditions: (1) during severe constriction of the renal artery, (2) after release of a clamp which has occluded the renal artery for more than 2 hours and (3) during recovery of the renal vascular bed from severe vasoconstriction produced by stimulation of the renal nerve or by vasoconstrictor substances.[55] India ink injected intravascularly appeared for the most part only in the cortex of kidneys during such conditions. This pattern of blood flow through the kidney is probably secondary to a marked reduction of renal blood flow which becomes insufficient to perfuse the more distally located tissues, as the medulla. It is to be noted, however, that under conditions of severe reduction of renal blood flow, most of the blood does go to the renal tissue that is highly susceptible to ischemia (the cortex), with only little, if any, going to the medulla, which apparently is more resistant to ischemia. Other investigators have also observed that when renal blood flow and pressure are severely reduced, the distribution of blood through the kidney is largely limited to the cortex, especially to the juxtamedullary region.[56-58] We have observed that often the medulla appears congested under the conditions in which India ink was found in the renal cortex only. Consequently, the presence of a congested medulla does not mean that an increased flow of blood or a shunting of blood through the medulla has occurred, an interpretation given in some reports.

There is evidence suggesting that blood flow is often best preserved through the juxtamedullary region during conditions in which blood flow to the kidneys is severely restricted. The vessels in this region were often hyperemic and the lesions were frequently much less severe in this region than in more peripheral parts of the cortex of kidneys following a prolonged period of hypotension. The cortex adjacent to the juxtamedullary region was sometimes completely destroyed whereas the juxtamedullary region itself showed only moderate tubular degeneration with little or no change in the glomeruli. In fact, in several instances the cortex superficial to and the medulla below this area were necrotic. Darmady,[59] Maegraith and Findlay,[45] and McManus[26] have noted similar histologic changes in human kidneys in cases of acute renal insufficiency. Ash[60] also observed this feature in bilateral cortical necrosis of human kidneys.

Vascular congestion of the medulla was frequently observed in the hypotension kidneys. Renal medullary congestion has been reported in man in shock.[61, 62] As already suggested, this finding, however, does not necessarily

indicate an increased flow of blood through the medulla nor does it indicate a deviation of blood away from the cortex to the medulla. Frequently ink injected intravascularly failed to appear in a congested medulla although it appeared in all or in part of the renal cortex. Ink sometimes failed to perfuse the congested medulla even 24 hours after the release of the clamp which had occluded the renal artery for 2 to 4 hours, although ink did perfuse the cortex.

It is difficult to state whether or not the renal lesions found in prolonged hypotension were specifically associated with shock. The lesions occurred in animals that never showed evidence of irreversible shock, as defined by Wiggers.[31] However, in animals which did go into irreversible shock, the lesions were severe and extensive, if the hypotension lasted more than 6 hours. That hypotension is not an absolute necessity for the pathogenesis of these lesions is indicated by the fact that they occur when large doses of epinephrine are given. In these latter experiments the mean arterial blood pressure of the animal usually did not fall until the last 20 to 30 minutes preceding death. The necessary conditions for the production of the lesions appear to be those which cause, over a period of several hours, a greatly diminished pressure and flow of blood in the renal vascular bed with or without a reduction in systemic blood pressure. Renal vasoconstriction probably plays an important role in producing the decrease in intrarenal blood flow.

Sudden severe hemorrhage in the dog does not cause significant renal damage or lesions such as are found with prolonged hypotension.[14] We removed 35 to 40 per cent of the estimated blood volume of 3 dogs by rapid bleeding from the carotid artery, but the kidneys contained only minimal scattered degenerative changes in the tubules when removed 4 to 5 days later. Oliguria did occur after the hemorrhage and the specific gravity of the urine remained high. Netravisesh and White[63] have shown that renal clearances return to normal within 48 hours after the last bleeding in dogs which were bled on 3 or 4 successive days, the blood loss totaling 5.7 to 5.9 per cent of the body weight. Anemia alone does not alter renal function appreciably although changes in renal blood flow and glomerular filtration rate occur as a result of changes in corpuscular volume percentage (hematocrit) and in blood volume.[64, 65] In man, sudden massive hemorrhage treated rapidly by transfusion does not cause renal insufficiency.[66] Evidently the hypotension must be prolonged before significant renal lesions occur.

Renal lesions similar to those found in our study of prolonged hypotension have been reported after cold injury to an extremity,[67] injections of epinephrine,[53] injections of vasopressin,[54] and injections of bacterial toxins.[38, 68-74] It is probable that the lesions produced by these varied procedures are also secondary to some type of vascular occlusion or ischemia of the renal cortex. According to De Navasquez[70] the lesions associated with the injection of bacterial toxins result from acute necrosis of the media of interlobular arteries and afferent arterioles in the peripheral cortex. But Byrom[54] considered that vasoconstriction was the cause of renal lesions that

followed the administration of vasopressin, and that medial necrosis of the arterial vessels, seen especially in infarcts, was due to prolonged spasm of the artery. Our findings support this latter concept.

The renal lesions observed in the kidneys in prolonged hypotension are also similar to those reported in man after severe shock and severe hemorrhage uncomplicated by hemoglobinemia or myoglobinemia.[20, 26, 46, 62, 75-77] The lesions seen in the human kidney include infarcts and bilateral cortical necrosis. The pathologic descriptions found in literature on bilateral cortical necrosis in man agree with those found when this entity occurred in our dogs.[48, 60] Bell[18] has stated that there is demonstrable vasospasm in renal arterioles in human bilateral cortical necrosis.

Certain areas in the renal lesions produced by prolonged hypotension may resemble microscopically those described for so-called lower nephron nephrosis, but this was not a striking feature in our studies and the severest damage was actually in the proximal tubules. Casts were more prominent in the lower nephron and collecting tubules of kidneys removed 5 days or more after the termination of hypotension. These casts consisted of hyaline material and also of a lighter pink-staining material. The associated tubules frequently showed degenerative changes. The hyaline material evidently originated from proximal tubules, for when these tubules degenerated the cells characteristically assumed a hyaline appearance. Furthermore, casts of this material in the distal tubules sometimes still contained degenerating nuclei. Also, these hyaline casts were found in the medulla only immediately below areas of the cortex in which hyaline degeneration of the proximal tubules was present. Other workers previously considered that the hyaline casts in the distal tubules might have originated from degenerating proximal tubules.[78, 79] Whether cast formation in the lower nephron several days after the period of hypotension is important in producing or exaggerating renal insufficiency cannot be stated. The predominant defect, however, is the destruction of tubular cells.

The renal lesions produced by hypotension are distinct from those seen after hemoglobinuric nephrosis or conditions in which the primary lesion consists of degeneration of the distal nephron with or without obstruction of these tubules by casts (transfusion kidney, cholemic nephrosis, sodium urate nephropathy, and so forth).[23, 25, 27, 76, 80] The last-mentioned entity has been also termed "lower nephron nephrosis," but this term has come to be used uncritically to include all cases of acute renal tubular degeneration or necrosis. We have studied the renal lesions produced in rats and dogs by the intravenous injection of hemolyzed homologous stroma-free red blood cells following severe water restriction. The degenerative changes associated with hemoglobinemia involve the entire tubule although they are more severe in the ascending limb of the loop of Henle and in the distal convoluted tubule and collecting tubule.[25] Grossly, the parts of the dog's and rat's kidneys involved by the pigment are black, a finding not observed in hypotension kidneys. Infarcts and medullary and juxtamedullary congestion which resulted from prolonged hypotension were not observed in hemoglobinuric nephrosis. Pigment casts were found in

hemoglobinuric nephrosis but were not present in kidneys after prolonged hypotension. The juxtamedullary region was often the part of the kidney in which the changes were most severe in hemoglobinuric nephrosis, whereas this region of the kidney was frequently the least involved after prolonged hypotension. That the renal lesions of shock and those of distal tubular degeneration and obstruction are different is emphasized by Allen,[76] Davis,[81] and Bywaters.[82]

Some of the confusion regarding the histologic picture of the renal lesions of shock and those of so-called lower nephron nephrosis may be due to the failure to distinguish between the lesions produced by shock or prolonged hypotension alone and those produced by excretion of pigment during conditions predisposing to the formation of casts but uncomplicated by shock or hypotension. Thus, some workers have maintained that the renal damage produced by shock is most pronounced in the proximal convoluted tubule while others consider that it is most pronounced in the lower nephron. From a comparison of renal lesions produced by prolonged hypotension and shock and those seen in hemoglobinuric nephrosis, we conclude that the lesions of hypotension or shock do frequently involve the proximal convoluted tubule most severely whereas the lesions of hemoglobinuric nephrosis involve the distal tubule (loop of Henle, distal convoluted tubule, and collecting tubule) most severely and consist of degenerative changes with or without the associated formation of casts in the tubules. However, in both conditions degeneration is present in all segments of the tubules to nearly the same degree of severity, so that localization of the most severe degree of damage to any segment of the tubule really is not very important. We believe that the lesions produced by shock or hypotension are due to prolonged ischemia while those of pigment nephropathies are due to tubular obstruction plus damage to associated tubular cells. The pathogenesis of the degenerative lesions in the cells of the tubules in pigment nephropathies is not entirely clear.

We should like to point out the fact that opinion varies as to the causal relationship between tubular obstruction by casts and degenerative tubular changes, and also as to which is most important in causing renal insufficiency. Some believe that tubular degeneration is most important.[83] However, in some of the kidneys that we studied, the areas of tubular degeneration were small and focal, but at least one pigment cast could be found in a distal tubule in the involved areas whereas pigment casts were not seen in the surrounding normal tissue. Thus, it is conceivable that cellular damage is related to the formation of the casts. Rather[84] in studying the effect of human crystalline hemoglobin in the rat found that damage to renal epithelium did not occur until large amounts of pigment were present in the lumen of the tubules.

Shock may be associated with conditions which produce pigment nephropathies. Therefore, it is conceivable that the renal lesions seen in some clinical cases may actually be a combination of those due to the pigment or injurious substance and those secondary to ischemia produced by shock or prolonged hypotension. Hemoglobinemia per se in the experimental animal or

man does not result in significant renal dysfuction but when associated with shock or hypotension the clinical and pathologic changes of hemoglobinuric nephrosis may occur. This suggests that renal lesions seen clinically may be due to both shock or hypotension and the excretion of pigment.[85, 86] The fact that medullary and juxtamedullary congestion with a pale cortex similar to that observed in our study of the kidney after prolonged hypotension has been reported in man in acute renal insufficiency, in blackwater fever, in the crush syndrome, and in transfusion kidney, supports the view that renal damage due both to renal ischemia and to pigments can occur.[23, 45, 87-89] Certainly in the crush syndrome, shock is frequently a factor in addition to the myoglobin nephropathy. Many patients with the crush syndrome have centrolobular necrosis of the liver, such as is found in shock.[42] Some investigators consider renal ischemia to be the most important factor in the pathogenesis of the crush syndrome,[10, 59, 61, 90, 91] while others consider tubular obstruction by casts as the important factor.[25, 30, 82, 84, 85, 92, 93] Both may well be important, that is, the ischemia produces some renal damage and decrease of the glomerular filtrate necessary for the formation of pigment casts which in turn is associated with additional tubular degeneration. Reports of clinical investigations of pigment nephropathies give conflicting conclusions regarding the importance of shock in the pathogenesis of this condition, but some workers believe shock usually occurs.[23, 26, 94] Experimentally, pigment nephropathy can be produced in the presence of water deprivation. Therefore, severe shock and hypotension are not the only conditions which facilitate the formation of pigment casts and associated renal damage during the excretion of pigment.[85, 92] Thus, dehydration also is one of the conditions which predispose to the development of pigment nephropathies. Consequently, the renal lesions of pigment nephropathies may or may not be complicated by renal damage due to shock or prolonged hypotension, that is, to renal ischemia.

Our study does emphasize that although at least the excretory phase of renal function is reduced nearly to zero during severe hypotension, it recovers rapidly after restoration of the blood pressure to normal. Inasmuch as the function of the kidney is largely dependent on the pressure and flow of blood through it, these alterations in function during hypotension are understandable. However, if the hypotension is maintained sufficiently long, persistent degenerative changes in the kidney occur and complete recovery of function may not take place subsequent to the restoration of blood pressure.

Even though the renal lesions produced in our study were frequently extensive, they usually were not sufficient to produce fatal renal failure or even clinically significant renal insufficiency. Studies of renal function in man during and after shock have also indicated that severe persistent renal dysfunction following recovery from shock is apparently not common.[1, 2] Uncomplicated major abdominal operations do not produce significant renal dysfunction.[95] It must be remembered that more than three-fourths of the renal substance must be removed before death from renal insufficiency occurs in dogs.[96-98] Furthermore, the renal artery of the remaining kidney in the dog, after the other has been removed, must be occluded for 4 hours or more

before fatal renal insufficiency occurs.[22, 99] Also, the amount of functioning renal parenchyma must be reduced to about one-third of normal before the various metabolites normally excreted by the kidney increase in the blood.[18, 100, 101] In the hypotension kidneys, large areas of renal substance often appeared completely normal when examined immediately, or as long as 11 days after, the termination of a period of hypotension lasting 24 hours during which the mean arterial pressure was kept at 70 mm. Hg.

A striking feature which often follows resection of large amounts of renal tissue is that of polyuria.[96, 98] Polyuria was also observed in some dogs following a period of hypotension. The kidneys of such dogs usually were characterized by scattered cortical lesions with normal tissue between the lesions. As a result of the decrease in functioning renal tissue, the remaining nephrons evidently eliminate the substances demanding excretion, but a volume of urine larger than usual is necessary. To produce anuria due primarily to renal destruction in the dog, the loss of functioning renal tissue must be very extensive.

It has been a common experience that renal failure in the dog is difficult to produce experimentally by either shock or hemoglobin.[9-13] Because of this fact some investigators believe that the dog's kidney is less susceptible than the human kidney to damage from shock.[9, 11, 13] Selkurt[7] did find mild diffuse cloudy swelling and hydropic degeneration of tubules following severe hypotension of about 3 hours' duration produced by hemorrhage in dogs. As already indicated, although pigments such as hemoglobin and myoglobin are not innocuous, it is difficult to produce primary renal failure by the intravascular injections of these substances in normal animals. Conditions predisposing to the formation of casts must pre-exist.[61, 85, 86, 102-108] Thus, although it is not easy to produce acute renal failure or renal lesions by hypotension or shock or with such pigments as hemoglobin or myoglobin, under proper conditions severe renal lesions do occur. The renal lesions so produced can be sufficiently extensive to cause death from acute renal failure.

Although reduction in renal clearances occurred after prolonged hypotension, elevation of the plasma values for nonprotein nitrogen was not very striking unless nearly all cortical renal tissue of the dog was destroyed. Evidently hypotension did decrease the function of sufficient amounts of renal tissue to cause decrease of clearances but enough functioning renal tissue remained to remove most of the substances normally excreted by the kidney. Functional studies also showed that the kidney usually regained its function rapidly. However, in 1 dog the cortical tissue of both kidneys was nearly completely destroyed. The clearances on this dog were markedly reduced, the plasma values for nonprotein nitrogen continued to rise, and death appeared imminent when the experiment was terminated on the fifth day after the period of hypotension.

Important additional factors, especially depletion of water and electrolytes, frequently operate in clinical cases of acute renal insufficiency. A feature of salt depletion is the rise in blood urea, as a number of investigators have emphasized.[109-112] Foy and co-workers[90] emphasized the importance of

this factor in acute renal insufficiency, both in predisposing to pigment nephropathy and in prolonging anuria and uremia after renal damage has occurred. Water intoxication is relatively easy to produce in salt-depleted animals.[113] Thus, extrarenal factors also must be considered in the pathogenesis of clinical acute renal insufficiency.

The following classification of the more common causes of acute renal insufficiency which may be seen in surgical patients appears useful (pre-existing renal disease and the complicating renal infections are excluded):

1. The concomitant presence of hypotension. The disturbances of renal function are reversible provided that the hypotension is not prolonged.
2. Severe renal damage as a sequela of prolonged hypotension or shock. Prolonged renal ischemia plays a major role in producing renal damage.
3. Acute tubular degeneration associated with obstructive cast formation by certain specific substances. This apparently occurs only under conditions in which the glomerular filtrate is markedly reduced. This category includes hemoglobinuric nephrosis, myoglobinuric nephrosis, certain sulfonamide nephroses, cholemic nephrosis, and so forth.
4. A combination of categories 2 and 3. This is seen in the crush syndrome.
5. Severe depletion of water or electrolytes (especially sodium chloride), or both. This is frequently seen clinically. It may be associated with categories 2, 3, and 4.
6. Obstruction of extrarenal urinary passages.
7. Occlusion of renal vessels.
8. Toxic chemical agents such as certain sulfonamides.

Our study and those of other investigators have demonstrated the importance of eliminating shock and hypotension in preventing acute renal failure. This is even more important in the presence of hemoglobinemia or myoglobinemia. It is of great importance to ensure that the volume of urine is high enough to prevent the occurrence of pigment nephropathy.

## SUMMARY AND CONCLUSIONS

1. Prolonged experimental hypotension produced in dogs by bleeding into a reservoir until a mean arterial blood pressure of 70 mm. Hg was attained and maintained at this level for periods ranging up to 26 hours was found to cause marked degenerative changes in the kidney. The severity and extent of renal involvement varied from focal areas of mild degenerative changes in tubules to bilateral cortical necrosis. Frequently, cortical infarcts were found. In general, hypotension had to be maintained for more than 6 hours before significant lesions occurred.

2. Although the renal lesions were often extensive and caused marked decreases in renal clearance and elevation of plasma values for nonprotein nitrogen to 110 mg. per 100 c.c., the renal damage usually was not sufficient to

produce death from renal failure. Evidently before death primarily from acute renal insufficiency occurs in the dog, nearly complete destruction of functioning renal tissue must occur.

3. The lesions were usually restricted to the cortex. Medullary congestion frequently occurred. Evidence indicated that blood flow is best maintained through the juxtamedullary region during the condition studied, but a deviation of blood flow through the medulla as postulated by Trueta and his associates[38] could not be confirmed. It is believed that the lesions result from renal vasoconstriction plus the prolonged decrease in blood pressure and flow to the kidney. This is independent of renal innervation. Renal lesions similar in all respects to those following a prolonged period of hypotension occurred after occlusion of a renal artery for 3 to 4 hours or after injection of large doses of epinephrine. These findings support the view that renal lesions from hypotension are due to prolonged ischemia. The presence of a congested medulla does not necessarily indicate that an increased flow of blood is occurring through the medulla. Actually, very little blood may be flowing through a congested medulla—it is simply static.

4. The lesions produced in hypotension are distinguished from those seen in hemoglobinuric nephrosis, in the so-called lower nephron nephrosis, and in conditons in which acute renal tubular degeneration is associated with pigment casts. As a result of hypotension all segments of the tubule are involved by degenerative changes, which are most severe in the proximal convoluted tubules, and pigment casts are essentially absent. In hemoglobinuric nephrosis or so-called lower nephron nephrosis the degenerative changes also involve all segments of tubules but appear to be maximal in the distal nephron (ascending limb of the loop of Henle, distal convoluted tubule, and collecting tubule) in the presence of varied numbers of pigment casts. However, in kidneys examined 5 or more days after the period of hypotension some areas fitting descriptions of so-called lower nephron nephrosis are seen due to the presence in the lower nephron of dark pink-staining casts of hyaline material, considered to have originated from degenerating proximal tubules, and casts of a lighter-staining pink material. Some associated distal tubules also showed vacuolar degeneration. However, the most severe injury observed in our studies was located in proximal convoluted tubules. Renal ischemia may occur concomitantly as pigment nephropathy is produced, and may be an important factor in the pathogenesis of the latter condition, thus making a combination of features of the two conditions in some cases possible (as in the crush syndrome).

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# ACUTE CHOLECYSTITIS

## Its Early Surgical Treatment; Report of 108 Cases

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OUR INTEREST in the early surgical treatment of acute cholecystitis was aroused in 1943 after the unfortunate experience of watching the gall bladders of two patients perforate while under observation in the hospital.[1] In changing our attitude toward one of early surgical intervention, several problems appeared. Obviously all patients with acute gall bladder colic do not need emergency surgery. We wished, therefore, to find some means of avoiding unnecessary emergency surgery and still avoiding perforation. We hoped also that it would prove possible in our hands to complete, with a high degree of regularity, all necessary biliary tract surgery in the acute phase of the disease. It is perhaps unnecessary to state that mortality and morbidity rates should prove acceptable.

One hundred eight patients with acute cholecystitis have been treated surgically. The results of this experience, together with some mistakes in diagnosis, have now been studied and constitute the basis for this paper.

### ANALYSIS OF MATERIAL

*Incidence.*—During a nine-year period, from Jan. 1, 1942, to Dec. 31, 1950, 804 patients were treated surgically for nonmalignant disease of the biliary tract. Of this group, 108 cases were clinically and pathologically acute. The acute cases thus constituted 13 per cent of the total group. It is interesting, however, that 22.4 per cent of the 274 patients treated surgically in the last two years were operated upon in the acute phase. This we interpreted as indicating an increasing satisfaction on the part of the surgical staff with earlier operation. The age of patients in the acute series ranged between 19 and 86. Thirty-eight per cent of them were men and 62 per cent women.

*Duration of Symptoms.*—The symptoms had been present for an average of 53 hours on admission to the hospital. Many patients were admitted within a few hours after the first pain but, in one instance, an interval of 12 days had elapsed from the onset of the pain to admission to the hospital. Sixty-three and one-half per cent gave a history of previous attacks.

*White Blood Cell Count.*—The leukocyte count ranged between 6,000 and 29,200 cells per cubic millimeter of blood. The average count was 15,100. It was normal (below 10,000) in 16.6 per cent. The polymorphonuclear count, however, was elevated in 89.5 per cent, and, in over 50 per cent, band forms were present in numbers greater than 5. In all of these patients in whom the total white blood count was below 10,000, pathologic examination of the specimens

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revealed varying degrees of acute cholecystitis. In several of these, acute hemorrhagic cholecystitis and gangrene were present. This conforms to the statements seen in the literature that a normal white blood cell count is no guarantee that serious gall bladder disease is not present.

*Treatment.*—Cholecystectomy was employed in 105, or 97 per cent, of the 108 cases. Thus, in only three cases in the series were technical difficulties such, or the condition of the patient such, as to require a simpler procedure. This ratio seems high as compared with other reported series. It no doubt reflects the tendency to operate on patients early in the course of their disease. The common duct was explored in 13 cases, or 12 per cent. Stones were recovered in 5.

*Mortality and Morbidity.*—One death was recorded among the 108 patients operated upon in the acute phase. Thus, the mortality rate was 0.92 per cent. This compares favorably with the mortality rate of 1 per cent in 696 nonacute cases. The postoperative hospitalization period averaged 9½ days.

Perforation was present in 11 cases, or 10 per cent. As in the previous report, we could find no correlation between the duration of symptoms and perforation. One patient's gall bladder perforated only 48 hours after the onset of symptoms. In another instance, 16 days had elapsed between onset of symptoms and operation, the patient having remained at home for 12 days before seeking relief. Even so, the gall bladder in this patient was not perforated, although it was pathologically acute.

The postoperative course was considered uneventful in 82 of the 108 patients, or 74 per cent. Of the remaining patients, disorientation was recorded in 3, cardiac decompensation in 4, wound infection in 5, hepatitis and cholangitis in 1, atelectasis in 1, wound separation in 3, in 2 of which secondary closure was necessary. Oliguria was present in 3 patients and thrombophlebitis in 1. Subhepatic abscess was present in 2 patients, 1 of whom required drainage.

We have discontinued the use of oxycel sponges to control bleeding in the gall bladder bed, since this was thought to be responsible for the development of subhepatic abscesses in the 2 cases cited above.

The 1 patient in the series who died was a patient 56 years of age, who was admitted with a 12 hour history of acute right upper abdominal pain which subsided within 24 hours after admission to the hospital. She was discharged in good condition only to return 3 days later with another attack. This time moderate tenderness was present in the right upper quadrant. No mass was felt. White blood cells numbered 21,400 with 86 per cent polymorphonuclears. She was operated upon 6 hours after this admission and an acutely inflamed gall bladder was found and removed without difficulty. Marked hypotension and oliguria were present for the first 7 postoperative days. She then developed chills and fever, and a diagnosis of septicemia was made. *Escherichia coli* was cultured from the blood stream on several occasions. Many antibiotic agents were given, none of which had any effect on the course of the septicemia, and she died on the eightieth postoperative day with terminal cardiac decompensation. Autopsy revealed multiple abscesses in the liver, spleen, kidneys,

and lungs. A small abscess was present in the gall bladder bed. It was not clear where this septicemia originated, although it was presumed to have been from the gall bladder area.

During the entire nine-year period, the hospital records indicate that in only 1 instance was recommended surgery refused for acute cholecystitis. This was an 86-year-old woman who developed severe upper abdominal pain 6 days after having undergone an internal fixation operation for intertrochanteric fracture of the right hip. At this time she was found to have marked right subcostal tenderness. A mass was not palpable. Leukocytes numbered 24,200 with 92 per cent segmented cells and 5 bands. Operation was advised but refused. She died 9 days later. Autopsy disclosed death to be due to bile peritonitis resulting from a perforated gall bladder.

*Mistaken Diagnosis.*—During the period under study 4 patients were operated upon under the mistaken diagnosis of acute cholecystitis. Two of these presented typical histories of right upper quadrant pain with tenderness and rigidity and an elevation of the white blood cell count to some degree. Operation disclosed acute hepatitis to be the cause of the symptoms. In one case the gall bladder was uninvolved, and in the other, stones were present. Biopsy of the liver in each case revealed an acute hepatitis, and in both of these cases recovery was prompt.

In the other 2 cases, myocardial infarction was the cause of the upper abdominal pain. Because we considered this of some importance in the management of patients with right upper abdominal pain, they are reported herewith in somewhat greater detail.

The first of these was a 75-year-old white woman with a cardiac history of considerable length, suggesting previous coronary occlusions with one probably occurring in the fairly recent past. On admission, she complained of acute retrosternal pain of several hours' duration and the clinical and electrocardiographic findings suggested the diagnosis of myocardial infarction. She also stated that she had had, during the past 3 months, several episodes of right subcostal pain, which she assumed to be due to gall bladder disease. The patient's condition was poor at the time of admission, but because of a persistent, apparently increasing abdominal pain and tenderness, surgical consultation was sought on the second day. Scout films of the abdomen had revealed gall stones in the region of the gall bladder, and a definite tenderness was present in the upper abdomen more marked in the right subcostal area. A mass was present in that area, presumed to be her gall bladder. Conservative management was advised because of the cardiac status of the patient, but the abdominal pain persisted and grew more troublesome. Finally, on the fifth day of admission, because of an increasingly tender mass in the right upper abdomen, a short subcostal incision was made under local anesthesia. The gall bladder was distended and contained stones but no acute inflammatory change was apparent. The liver was markedly enlarged and congested. Stones were removed and cholecystostomy was done. It was felt at the time of the operation

that the tenderness present had been due to an enlarged liver. The patient died the following day. Autopsy disclosed the cause of death to be extensive infarction of the left ventricle.

The other patient was a 78-year-old man who had previously enjoyed fairly good health except for the fact that he had noted some vague distress in his upper abdomen after eating greasy foods. This he had noted for approximately 2 years. Two days prior to admission he was seized with a severe right upper quadrant pain which radiated into his right substernal region. The pain had been constant for 2 days, though worse at intervals, and definitely cramping in nature. He had vomited several times a day prior to admission. The referring physician felt that he was suffering from either an acute gall bladder attack or an acute myocardial infarction. Physical examination disclosed an elderly white man obviously quite ill. The skin was moist and cool. No jaundice was present. The blood pressure was 120/50, pulse was 110 beats per minute, and of fairly good quality. Examination of the chest and heart revealed no abnormalities. The abdomen was not distended, but was remarkably tender over the right upper quadrant with moderate muscular rigidity. A mass was not felt. Examination of the blood disclosed the hemoglobin to be 10.4 Gm., red blood cells numbered 3,800,000, and leukocytes numbered 27,400, with 11 lymphocytes, 79 segmented cells, and 10 bands. A medical consultant examined the patient and obtained an electrocardiogram which showed evidence of a posterior wall infarction. The patient was watched at the bedside for several hours and during paroxysms of pain he held his hand over his right subcostal area as the place where the most severe pain was located. It was felt by the medical consultant as well as ourselves that whereas this patient had evidence of a posterior wall infarction, it could not be determined whether this was recent or old. We felt that he had an acute cholecystitis with impending perforation. Consequently, he was operated upon under local anesthesia at which time he was supported by a blood transfusion. Upon opening the peritoneal cavity the blood pressure dropped to dangerous levels so that the operation had to be discontinued for a few moments to allow him to become stabilized. Quick exploration revealed the liver to be normal in size and appearance. The gall bladder was thin walled and not inflamed. No stones were present. Palpation of the remainder of the abdominal contents revealed no cause for his acute abdominal symptoms. The wound was closed. The patient died the following day. Autopsy was not permitted. The cause of death was felt to be an acute myocardial infarction.

## DISCUSSION

*Etiology.*—Interesting studies on the etiology of cholecystitis have been reported by Womack and Haffner.[2] They doubted the frequency of primary bacterial invasion and presented experimental and clinical evidence to show that perhaps all forms of cholecystitis are primarily chemical. If an infectious process is present, it is secondary invasion of previously damaged tissue. Bile itself is the chemical responsible. They emphasized the following factors in the patho-

genesis of acute cholecystitis: (1) obstruction of the cystic duct, (2) action of bile on the gall bladder wall, and (3) occasional secondary bacterial invasion superimposed on chemically damaged tissue.

The cystic duct obstruction is due to stone in 95 per cent of cases. There then follows increased intravesicle pressure from secretion, exudation, and transudation and, finally, vascular interference with gangrene and perforation. Barksdale and Johnston[3] have postulated that nature will interrupt the cycle in 80 to 90 per cent by relieving the obstruction either by dislodgment of the stone back into the gall bladder or passing it into the common duct. Thus, in 10 to 20 per cent, surgical interference is absolutely necessary to avoid perforation. Goldman[4] reported on bacteriologic studies in 160 cases of acute gall bladder disease which lend further support to the theory that bacterial invasion is late and that initially the process is a chemical and/or a mechanical one. He found a low incidence of positive cultures in the first 3 days of the disease and a much higher incidence of positive cultures between the fourth and eighth days when the mortality rate was also highest.

*Incidence.*—It is difficult to account for the wide variation of incidence reported. Heuer[5] found acute cholecystitis in 23.5 per cent of 1,142 patients with gall bladder disease. Buxton, Ray, and Coller[6] found only 1.5 per cent in 7,019 cases. They take a more conservative attitude. These authors say that it is most likely that many more persons treated without operation might have shown acute cholecystitis had the gall bladders been removed and examined by a pathologist.

Dunphy and Ross[7] reported 649 cases of gall bladder disease. In 20 per cent, acute cholecystitis was present. This is from the Peter Bent Brigham Hospital where conservative management of acute cholecystitis was abandoned in 1935.

*Diagnosis and Treatment.*—While the diagnosis of acute cholecystic disease is seldom difficult it is often extremely difficult to know how far the disease has progressed. The characteristic colicky right upper abdominal pain is well known. Often the patient himself has made the diagnosis. In acute cholecystitis the pain is often sustained and unrelenting with radiation into the infrasternal region. The presence of a palpable tender mass in the right subcostal area in a patient acutely ill with colicky pain makes operative intervention mandatory as soon as proper preparation will permit. The opposite, however, is not true, namely, that conservative management is safe as long as the gall bladder is not palpable. This is in disagreement with Saint[8] who claims that if gangrene and impending perforation are present, one should always feel a distended tender mass. We have often encountered gangrenous gall bladders lying high, well up under the costal margin so that even under anesthesia, before making the incision, palpation of this distended viscus was not possible.

There seems to be no clinical or laboratory procedure at present that will tell the surgeon when gangrene is present and perforation impending. Best[9] reported 44 cases of acute gall bladder disease, one-half of which were treated by a conservative policy and the other by early surgical intervention, and he

stated "the lack of parallelism between the pathology and the laboratory-clinical findings is often more astounding in acute cholecystitis than in acute appendicitis."

Eliason and Stevens[10] reported on studies made at the gastrointestinal clinic at the University of Pennsylvania and found that biliary tract disease accounted for 40 per cent of all the cases seen there. Of these, 20 per cent were classified as the acute type. They feel that a true obstructive gall bladder disease of more than 2 days' duration will not subside under any therapy except surgery. The pathologic state of the gall bladder cannot safely be estimated by any physical or laboratory test; hence the danger of delay. They found in 135 cases that the white blood cell count was normal in 21 per cent. Temperature was normal in 23 per cent. No mass was palpable in 30 per cent. Even local tenderness was absent in 4 per cent.

McNealy[11] reported 500 cases of acute gall bladder disease seen at the Cook County Hospital. The white blood cell count was fairly consistently over 10,000 with perforation, gangrene, or empyema, but he states that one cannot rely upon it. He felt pain to be the most reliable sign. In only 29 per cent of these cases was a palpable mass present.

Wallace and Allen[12] reported on 2,273 operations for gall bladder disease during a ten-year period at the Massachusetts General Hospital. Of these, 18.2 per cent had acute gall bladder disease with a 6 per cent mortality. They stated that elevated temperature and white blood cell counts are unreliable guides for estimating the stage of the disease process. Of the patients in that series with gall bladders perforated, 30 probably perforated while under observation in the hospital!

Marshall[13] felt that abdominal signs and laboratory findings do not always give reliable indices to the severity of the inflammatory process in the gall bladder, and that watchful waiting may lead to complications. It is, therefore, the custom at the Lahey Clinic to regard every case of acute cholecystitis as an emergency and to operate as early as possible.

Cowley and Harkins,[14] in 25 cases of perforated gall bladder, found the white blood cell count in 24 per cent to be below 10,000. Tenderness was present in 100 per cent of cases, and a mass was present in only 24 per cent.

Thus, in advising operation on the patient with acute cholecystitis, fever, leukocytosis, increase in toxic form of white blood cells, and a palpable mass are valuable diagnostic aids *if present.* It is possible and fairly common to find a progressive acute process in the gall bladder in the absence of any or all of these.

The most reliable sign in our experience is persistent pain. If pain and tenderness disappear within 48 hours we feel it is usually safe to treat the patient conservatively so that more detailed diagnostic studies can be carried out and operation then recommended in a quiescent period.

The treatment of choice should be cholecystectomy with exploration of the common duct as indicated. This has been possible in a high percentage of our own cases, although it is agreed that cholecystectomy on the acutely in-

flamed gall bladder is often more difficult than on the nonacute type. Wide exposure is obtained through a subcostal incision. It is felt that gas-oxygen anesthesia administered through an endotracheal tube combined with intravenous Pentothal Sodium and curare offers the best anesthetic combination. The gall bladder is commonly found to be covered with edematous omentum which is easily separated with a gloved finger. It is almost always necessary to aspirate the gall bladder with a trocar, open the fundus widely and remove the stones, being especially careful to remove impacted stones where a proper exposure of the ducts is made easier. It is commonly possible to brush the edematous peritoneum-covered fatty tissues away from the ducts with gauze. Sometimes this is facilitated with the left forefinger inside the gall bladder. The only sure way to avoid injury to the common duct is to carefully expose it early in the course of dissection. Lahey's[15] suggestion of leaving the cystic duct intact until after the cystic artery has been divided and ligated is an excellent one that makes it possible to avoid tearing and retraction of the cystic artery from traction. The pericholecystic edema always present in acute cholecystic disease usually makes it easy to separate gently the gall bladder from its bed with a gloved finger. At times this is done before the cystic duct and the cystic artery are divided to facilitate exposure. All cases are drained with a single Penrose drain brought out through a stab wound to the right of the incision together with a T tube if it has been necessary to explore the common bile duct.

Cholecystostomy, however, remains as a definite procedure in the surgeon's armamentarium in the treatment of acute cholecystic disease. It is indicated in the severely toxic patient, especially an elderly patient in whom the disease has been present for some time. In this type of patient cholecystostomy may be a lifesaving procedure and can be done under local anesthesia.

*Common Duct Stones.*—It has occasionally been suggested that the very factor which usually produced acute gall bladder attacks, namely, impaction of a stone in the cystic duct, often precluded the possibility of common duct stones. Some men have stated, however, that common duct stones are found in approximately the same ratio in acute as in chronic cases. Jaundice may occur in acute cholecystitis in the absence of common duct stones so that the decision as to whether or not to explore the common duct may be a difficult one. Lester,[23] in 1947, reported 109 cases of acute cholecystitis seen at the New York Mount Sinai Hospital. Jaundice was present in 19, or 17 per cent. In only 6 cases was the jaundice due to common duct stones. He concluded that jaundice alone without dilatation of the common duct is not an indication for common duct exploration in the patient with acute cholecystitis.

Dunphy and Ross[7] reported 134 cases of acute cholecystitis seen at the Peter Bent Brigham Hospital during the seven-year period between 1941 and 1947. They state that jaundice may occur in acute cholecystitis without common duct stones, although they explored the common duct in 50 cases in the above series and recovered stones in 17 per cent. In our experience with the 108 acute cases reported here, the common duct was explored in 13, or 12 per

cent. Stones were recovered in 5. This percentage seems low in comparison with the experience of others, although, to our knowledge, no patient has needed reoperation for common duct stones. In several, however, there has been present so-called biliary dyskinesia without jaundice, which has been relieved by Best's biliary flush.[9] Jaundice was present in 6 patients in whom the common duct was not explored. In all of them the jaundice subsided promptly after surgery.

*Mortality Rates.*—The mortality rates reported following the early surgical treatment of acute gall bladder disease are almost uniformly slightly higher than the mortality rates reported following operations for chronic gall bladder disease. However, a considerable advantage to the patient would ensue if he were allowed the alternative of facing a 10 to 20 per cent possibility of perforation with its attending average mortality rate of 45 per cent (Marshall and Phillips).[16] More recently reported mortality figures show gratifying gains over past years. This is due not only to improvements in anesthesia and use of antibiotics, but also to the gradual realization by surgeons that early, prompt surgical intervention constitutes the ideal type of treatment when very little edema is present and technical difficulties are reduced to a minimum. Eliason and Stevens[10] in their 135 reported cases of acute cholecystic disease treated by early operation had only 2 deaths, a mortality rate of 1.5 per cent. Heuer was one of the first advocates of early surgical intervention, having seen Halsted remove an acute gall bladder in 1910. He was responsible for initiating a policy of early surgical intervention in cases of acute cholecystitis in the New York Hospital in 1932. From that time up to 1945, Glenn and Heuer[17] reported 527 cases treated with early surgery with 13 deaths, a mortality rate of 2.4 per cent. Cholecystectomy was employed in 87.4 per cent and cholecystostomy in the remainder. Gray and Lofgren[18] recently reported studies on acute cholecystitis from material seen at the Mayo Clinic. During an eight-year period cholecystectomy was done for 455 cases of acute cholecystitis with a mortality rate of 2.9 per cent. During the same period 103 patients with acute cholecystic disease were thought to be too ill for cholecystectomy and consequently were treated by cholecystostomy. This represented 18 per cent of the group. Mortality rates reported by others include McGuigan,[19] 5.6 per cent, McLannahan, Trout, and Weary,[20] 8.6 per cent, and Zollinger and Cutler,[21] 2.6 per cent.

The mortality rate in acute cholecystic disease appears to rise sharply in patients over 50 years of age. This is due to coexisting diseases of old age such as diabetes, high blood pressure, and arteriosclerosis which often complicates the problem considerably. There appears to be a greater tendency for elderly patients to procrastinate before seeking relief. Consequently, many of these patients are seen late in the course of the disease. Glenn[22] reported 93 cases of acute cholecystic disease in patients over 50 years of age. Sixty-nine were treated with cholecystectomy and the others by cholecystostomy. The over-all mortality rate was 6.4 per cent. McLannahan found that the mortality rate was 8 times greater in patients over 60 years of age.

## CONCLUSIONS

We believe that all patients with acute gall bladder pain should be admitted to the hospital for observation. If the symptoms subside within 48 hours, it is safe to carry out detailed studies and advise operation as indicated at a later date. The development of a tender, palpable gall bladder at any time makes delay extremely hazardous and in these patients operation should be carried out as soon as the necessary few hours have elapsed for proper preparation of the patient. A high leukocyte count, especially if accompanied by a toxic change in the differential count, is an important diagnostic aid. Leukocytosis and a palpable mass, however, may be absent fairly commonly with a dangerous, progressing gall bladder condition. In this latter group, the most helpful aid to us has been the persistence of severe, unrelenting pain. If this lasts beyond 48 hours, operation should be advised.

Except in the rare circumstance where thrombosis of the cystic artery has occurred, operation 48 hours after the onset of symptoms does not confront one with serious technical difficulties. It should be possible to do a cholecystectomy with exploration of the common duct as indicated in almost every case (97 per cent in this series). Cholecystostomy, however, should be employed in the occasional patient who is very ill, probably having had his disease for some time before being hospitalized.

The average postoperative hospitalization period of 9½ days for these 108 patients suggests an economic benefit as well. For if one assumed that all acute attacks would eventually subside without complications, the patient would still face the prospect of returning for surgery at a later date, in which event the total period of hospitalization would be approximately doubled.

In our experience with this small series of patients with acute cholecystitis, the condition most difficult to differentiate was acute myocardial infarction, and the ill-advised operations in the two cases cited may well have hastened the fatal outcome.

## SUMMARY

1. One hundred eight cases of acute cholecystitis are presented,* in all of which surgical treatment was carried out with one death, or a mortality rate of 0.92 per cent.

2. Some of the problems connected with the treatment of this disease are outlined. The reasons for advising early surgical intervention are presented.

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*Since submitting this paper for publication, we have seen forty-nine additional patients with acute cholecystitis who required surgical treatment according to the criteria set forth herein. Cholecystectomy was employed in all with no mortality.

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# THE EFFECT OF DOUBLE VAGOTOMY ON THE MOTOR ACTIVITY OF THE HUMAN GALL BLADDER

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IN MAMMALS it has long been recognized that vesicular bile is expelled by contraction of the gall bladder musculature. In the laboratory animals, numerous experiments have demonstrated that this musculature and the sphincters at the end of the common duct are under the control of nervous as well as hormonal mechanisms. For example, section of the right vagus in the cat (and to a lesser extent, section of the left vagus) markedly delays the evacuation of a gall bladder that is emptying under the stimulus of a fatty meal.[11] In man, however, the experimental evidence has suggested that much of the nervous control of the extrahepatic biliary system has been lost and that evacuation of bile depends primarily upon the humoral mechanism.[3] Even as late as 1947, Ivy[10] stated that "to the present time there is no evidence that nerves have any influence on the motor mechanism of the human biliary tract." Nevertheless, there are frequent statements in the literature that attribute faulty function of the gall bladder and spasm of the sphincter of Oddi to nervous imbalance.[6, 8, 14]

Because of the complexity of the anatomy in this region and the difficulty of carrying out controlled experiments in the human being, the relative importance of the nervous system in regulation of the biliary tract has not been adequately worked out for man. Recently, however, the revival of popularity of bilateral vagus nerve section in the treatment of duodenal ulcer has presented an ideal opportunity for directly testing the role of the nervous system.

## METHOD

The subjects used were adult men, healthy in all respects except for a long-standing duodenal ulcer which was poorly controlled by medical means.

The normal rate of emptying of the gall bladder for each patient was established preoperatively. The gall bladder was visualized with 12 tablets of an oral gall bladder dye (Priodax). It was felt that this double dose of dye resulted in a much sharper gall bladder shadow, thus facilitating the tracing of outlines of the viscus on x-ray films.

About 15 hours after the administration of the Priodax, the presence of a satisfactory shadow was established by means of a preliminary film. Having thus accurately located the gall bladder, the x-ray field was "coned down" over this viscus. A modified "Boyden meal" of three egg yolks and an equal amount of cream was then injected through the nasal tube. At the time the

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injection was started, a zero film was exposed and subsequent pictures were taken at 2, 4, 8, 12, 16, 20, 25, 30, 35, 40 and 45 minutes after the test meal. The outlines of the gall bladder shadows obtained on these films were then traced on a good quality tracing paper and the volume for each was calculated from a chart prepared by Siffert[16] based on a method described by Boyden.[1]

Following the vagotomy, a period of at least three months was allowed to elapse before the postoperative rate of evacuation of the gall bladder was determined. It was felt that this should be a sufficient length of time for the subject to recover from the "shock" of the operation. Any difference between the postoperative and preoperative rates of evacuation which exceeded the return to a normal rate of emptying might then be attributed to section of the vagus nerves.

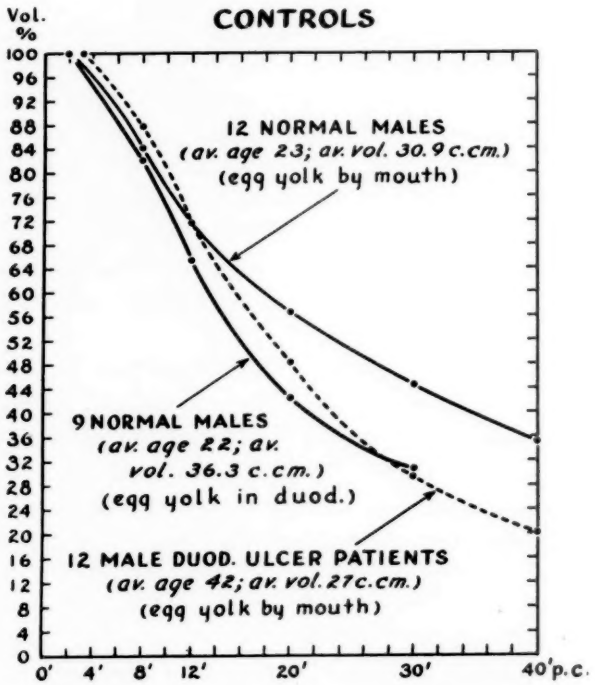


Fig. 1.

The decision as to whether the vagotomy was complete or incomplete was based on the Hollander insulin gastric secretion test.[9] Following injection of insulin, the titer of acid increases in the stomach through a reflex mediated by the vagus nerves. Hence if the nerves are completely severed there should be no appreciable rise in acid following vagotomy.

Finally, in interpreting these findings it is necessary to keep in mind certain factors especially pertinent to this study which govern the evacuation of the human gall bladder. First, it should be noted that in man the rate of emptying is not affected by variations in the fasting size of individual gall bladders.[1] Thus it makes little difference whether some patients have larger gall bladders than others. Second, age and sex do affect the rate. Since all

our patients were men, only the former will be considered. On the average, older men have a somewhat faster rate than younger men.[4] Third, evacuation of this organ is markedly accelerated in peptic ulcer patients.[5] Accordingly, the curves of evacuation in the preoperative series will be steeper than in normal men. (See Fig. 1.) Fourth, it makes considerable difference whether the motor meal is given by mouth or injected directly into the duodenum. For it has been shown[2] that as little as 10 c.c. of egg yolk introduced directly into the duodenum will produce a maximum contraction of the human gall bladder (Fig. 1). Also, it is known that in certain laboratory animals the time required for the initial passage of food into the duodenum is decreased by double vagotomy.[12] From these observations it is obvious that the initial period of discharge from the stomach is a more significant factor for this study than the time necessary for complete emptying of the stomach which, as is well known, is frequently prolonged in vagotomized patients. Therefore, an attempt was made to pass a mercury-weighted Levine tube into the duodenum on the afternoon before the day when the study was to be carried out. However, in certain ulcer cases and vagotomized patients this was found to be quite difficult. Accordingly, unless otherwise stated, the motor meal was injected into the stomach.

## OBSERVATIONS

From eleven out of thirteen male patients studied over a three-year period, satisfactory series of cholecystograms were obtained both before and after the operations for vagotomy. When all the data had been assembled it was found that the eleven series could be divided naturally into two groups, hereafter to be designated Groups A and B.

*Group A. Incomplete Vagotomies.*—The first of these (Group A) may be described as a negative control group, for in four of five cases the Hollander test induced a rise in free hydrochloric acid varying from 28° to 40° following what must be designated as incomplete vagotomies. In the fifth one (Case 10) there was a rise of only 20° within two and a half months of the operation. Possibly, this is a borderline case, for it is generally considered that a rise of less than 20° is not significant. These five will be discussed individually because they illustrate some of the factors involved in this study.

In Case 11 (Fig. 2, *A*), preoperative and postoperative curves were essentially the same. The gall bladder went into tonus 2 minutes after the standard meal, then relaxed for 2 to 4 minutes, then started its main phase of evacuation. Taking the 2 minute readings as unity (100 per cent) the rate of discharge has been calculated, according to the method of Boyden,[1] and presented in the form of curves (*A'*) which register the diminishing volumes in per cent. In this series a postoperative rise of acid from 0° to 40°, following the Hollander test, indicates that the vagotomy was incomplete. Correspondingly, there is virtually no change in the rate of evacuation. The curves are unusually steep, even for ulcer patients and illustrate the variation in individual behaviour

which characterizes the evacuation of the gall bladder. It is only when the data are averaged from a group of patients that conclusions can be drawn about the mean rate of discharge under varying conditions.

In Case 13 (Fig. 2, *B* and *B'*) it was possible to introduce egg yolk directly into the duodenum. Four months postoperatively, the rate of evacuation was slightly less, but not statistically significant. The Hollander test indicated that vagotomy was incomplete.

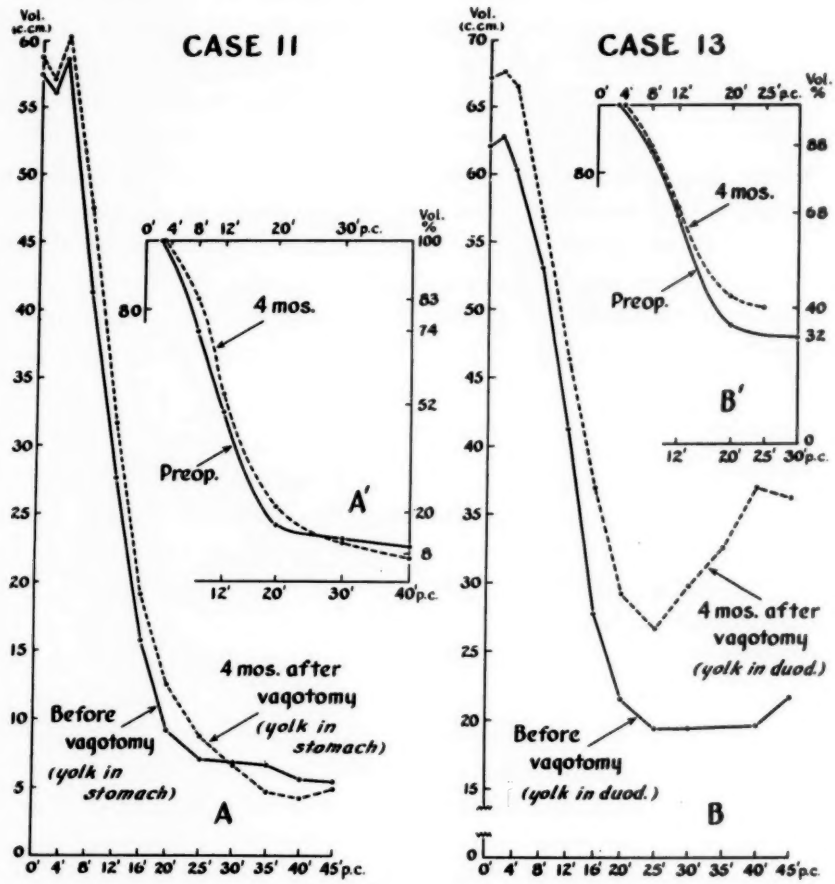


Fig. 2.

In Case 9 food was given preoperatively by mouth and postoperatively by duodenal tube (Fig. 3, *A*). The Hollander test indicated that vagotomy was incomplete. The postoperative curve was significantly faster, in accordance with the factor demonstrated in Fig. 1, namely, that food introduced directly into the duodenum accelerates evacuation of gall bladder bile. Case 12 (Patient H. A., age 36) was similar to Case 9.

In Case 10 a new factor has been introduced (Fig. 3, *B* and *B'*). For some unknown reason, the fasting volume of the gall bladder postoperatively was less than half that of the preoperative series. If it was due to the fact that the gall bladder had discharged some of its contents during fasting, that might

explain the slower rate of emptying, for an examination of any of the curves shows that toward the end of a phase of contraction the gall bladder slows down. On the other hand, the Hollander test showed a rise in free acid of from only 0° to 20°, so that it is possible that the slower postoperative rate was due to vagotomy. However, this case differs in another important respect from those in Group B, so that the weight of evidence is in favor of the first explanation.

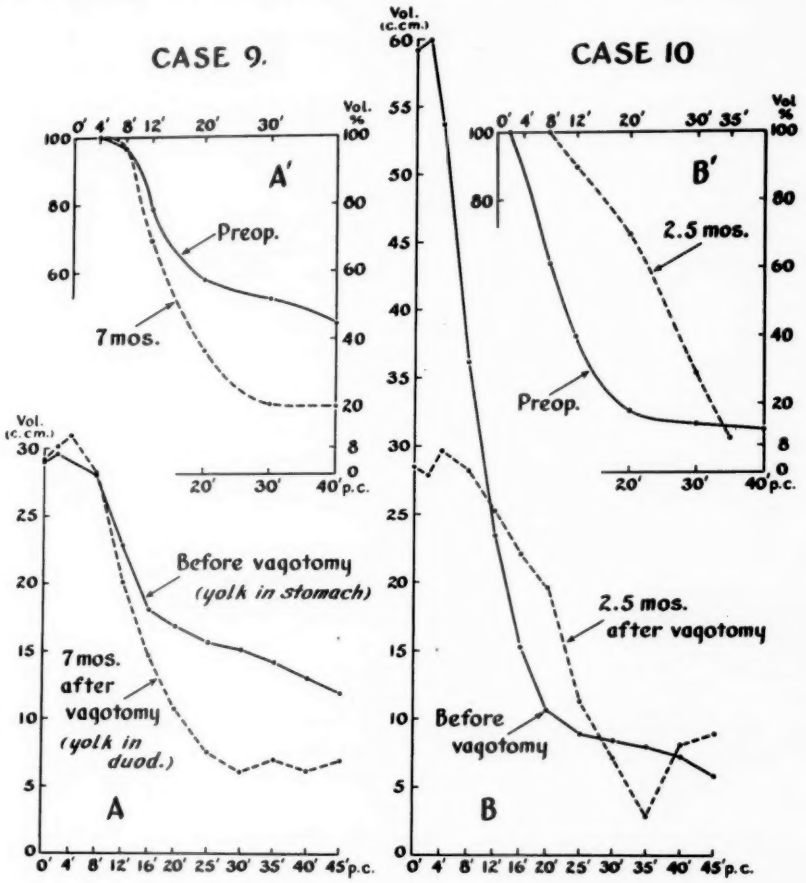


Fig. 3.

## *Summary of Group A*

CASE 9.—Preoperative Hollander test showed a rise in free HCl from 0° to 112°. Transthoracic vagotomy. Operation incomplete. At 2 weeks, a rise of 28° in free acid (10° to 38°); at 3.5 months, 40° (10° to 50°); at 7 months, the same rise. At 7 months, rate of evacuation of gall bladder was faster than preoperatively due to the fact that food was introduced directly into the duodenum.

CASE 10.—Preoperative rise of free acid from 10° to 100°. Transthoracic vagotomy. Operation incomplete? At 1 week, no rise in acid; at 2.5 months, a rise of free acid from 0° to 20°; rate of evacuation slower, possibly due to the fact that fasting volume of gall bladder was half as large as preoperatively.

CASE 11.—Preoperative rise of free acid, from 20° to 90°. Transthoracic vagotomy. Operation incomplete. At 2 weeks, a 40° rise (0° to 40°). At 4 months, rate of evacuation same as preoperatively.

CASE 12.—Preoperative rise of free acid, from 20° to 110°. Transthoracic vagotomy. Operation incomplete. At 11 days, a 40° rise (10° to 50°). At 5 months, rate of evacuation faster, due to duodenal intubation.

CASE 13.—Preoperative rise of free acid, from 60° to 130°. Transthoracic vagotomy. Operation incomplete. All 11 days, a 40° rise (20° to 60°); at 4 months, 34° (72° to 106°). At 4 months, rate of evacuation slower, but not significantly so.

*Group B. Complete Vagotomies.*—This group of six patients differed in three major respects from the patients described in Group A. First, the vagotomies appeared to be complete, for in four of the six patients there was no early rise in free HCl following the Hollander test, and in the remaining two (Case 4 and Case 5) only 8 degrees.*

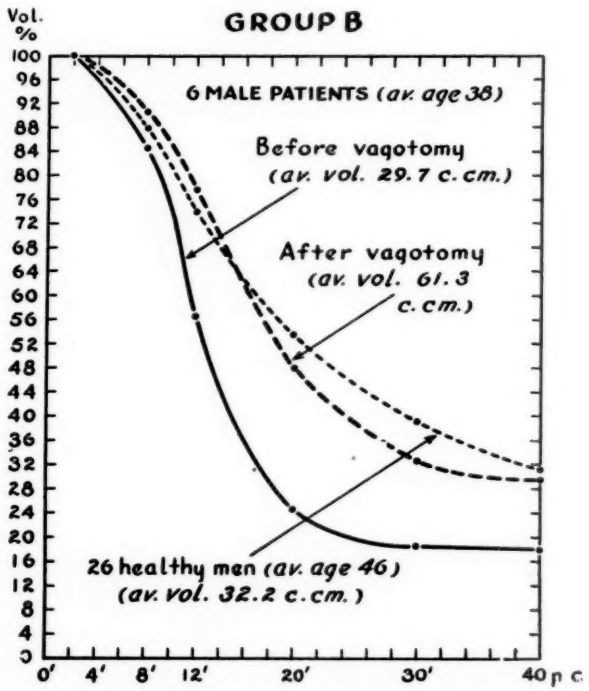


Fig. 4.

Second, in every case the rate of emptying of the gall bladder was significantly slower, postoperatively (see Table I). Even so, the postoperative rate returned merely to that of healthy men without ulcers (Fig. 4).

Third, in each case the fasting volume of the gall bladder was approximately doubled following complete vagotomy—an average of 61.3 c.c. as against 29.7 c.c. (Table I, column 3).

The most instructive cases of Group B are Cases 7 and 6. Accordingly these will be treated in some detail. Fig. 5, *A* shows the four successive series of cholecystograms of Case 7. From below upward the curves record the emptying of the gall bladder preoperatively, then at sixteen days, eight months

*It is to be regretted that in a number of instances the Hollander tests and cholecystographic series were not spaced more evenly, but the difficulties of getting patients to return for examination or of obtaining their cooperation were very great.

TABLE I. PATIENTS OF GROUP B WITH POSTOPERATIVELY DILATED GALL BLADDERS

| CASE | AGE | VOLUME OF GALL BLADDER, AND VOLUME PER CENT LOSS, AT INTERVALS AFTER FOOD | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | VOL. AT TIME OF TONUS* IN C.C. | 8 MINUTES | | 12 MINUTES | | 20 MINUTES | | 30 MINUTES | | 40 MINUTES | |
| | | | C.C. | PER CENT LOSS | C.C. | PER CENT LOSS | C.C. | PER CENT LOSS | C.C. | PER CENT LOSS | C.C. | PER CENT LOSS |
| | | | | | *Before Vagotomy* | | | | | | | |
| 1. (J. K.) | 53 | 38.5 | 32.0 | 16.9 | 24.5 | 36.4 | 13.0 | 66.2 | 8.4 | 78.2 | 10.1 | (78.2) |
| 3. (J. W.) | 28 | 36.9 | 31.2 | 15.5 | 22.0 | 40.5 | 8.2 | 77.8 | 4.0 | 89.2 | 6.0 | (89.2) |
| 4. (D. T.) | 28 | 23.6 | 21.9 | 7.2 | 15.5 | 34.3 | 3.2 | 86.5 | 4.8 | (86.5) | 5.1 | (86.5) |
| 5. (J. S.) | 60 | 27.1 | 20.3 | 25.1 | 12.8 | 52.8 | 7.0 | 74.2 | 5.8 | 78.6 | 7.2 | (78.6) |
| 6. (J. H.) | 32 | 29.0 | 27.2 | 6.2 | 10.7 | 63.0 | 4.3 | 85.2 | 3.9 | 86.6 | 8.4 | (86.6) |
| 7. (L. C.) | 29 | 22.9 | 18.0 | 21.4 | 14.9 | 34.9 | 8.2 | 64.2 | 7.4 | 67.7 | 6.1 | 73.4 |
| Totals | 230 | 178.0 | | 92.3 | | 261.9 | | 454.1 | | 486.8 | | 492.5 |
| Means | 38.3 | 29.7 | | 15.4 | | 43.6 | | 75.7 | | 81.1 | | 82.1 |
| Reciprocals | | | | 84.6 | | 56.4 | | 24.3 | | 18.9 | | 17.9 |
| | MONTHS POSTOP. | | | | | | | | | | | |
| | | | | | *After Vagotomy* | | | | | | | |
| 1. (J. K.) | 3 | 94.3 | 80.0 | 15.2 | 60.2 | 36.2 | 47.3 | 49.8 | 36.0 | 61.8 | 35.2† | 62.7 |
| 3. (J. W.) | 14 | 59.0 | 58.0 | 1.7 | 47.6 | 20.4 | 27.6 | 53.2 | 14.3 | 75.7 | 11.1† | 81.2 |
| 4. (D. T.) | 22 | 50.8 | 45.6 | 10.0 | 38.4 | 24.4 | 21.4 | 57.9 | 23.0 | 57.9 | 27.3 | 57.9 |
| 5. (J. S.) | 9 | 57.0 | 59.2 | 0.0 | 57.2 | 0.0 | 30.0 | 47.4 | 13.6 | 76.1 | 14.5 | (76.1) |
| 6. (J. H.) | 16.5 | 62.9 | 52.5 | 16.5 | 44.3 | 29.6 | 24.5 | 61.0 | 15.8 | 74.9 | 13.9† | 77.9 |
| 7. (L. C.) | 8 | 43.8 | 38.5 | 12.1 | 33.8 | 22.8 | 25.0 | 42.9 | 17.8 | 59.4 | 14.4 | 67.1 |
| Totals | | 367.8 | | 55.5 | | 133.4 | | 312.2 | | 405.8 | | 422.9 |
| Means | | 61.3 | | 9.2 | | 22.2 | | 52.0 | | 67.6 | | 70.5 |
| Reciprocals | | | | 90.8 | | 77.8 | | 48.0 | | 32.4 | | 29.5 |

*Considered as 100 per cent.
†At 25 minutes.

and fourteen months postoperatively. The last three curves show progressive increase in the fasting volume of the gall bladder. At six months there had been no rise in free HCl following the Hollander test, and at thirteen months only 12°. Consequently, it is clear that virtually all vagal fibers have been severed. At *A'* (Fig. 5) the data have been calculated for comparison of rates of emptying—the volume at which each gall bladder came into tonus being considered as 100 per cent. Here it may be noted that the three postoperative curves are approximately alike and that they are significantly slower than the preoperative curve.

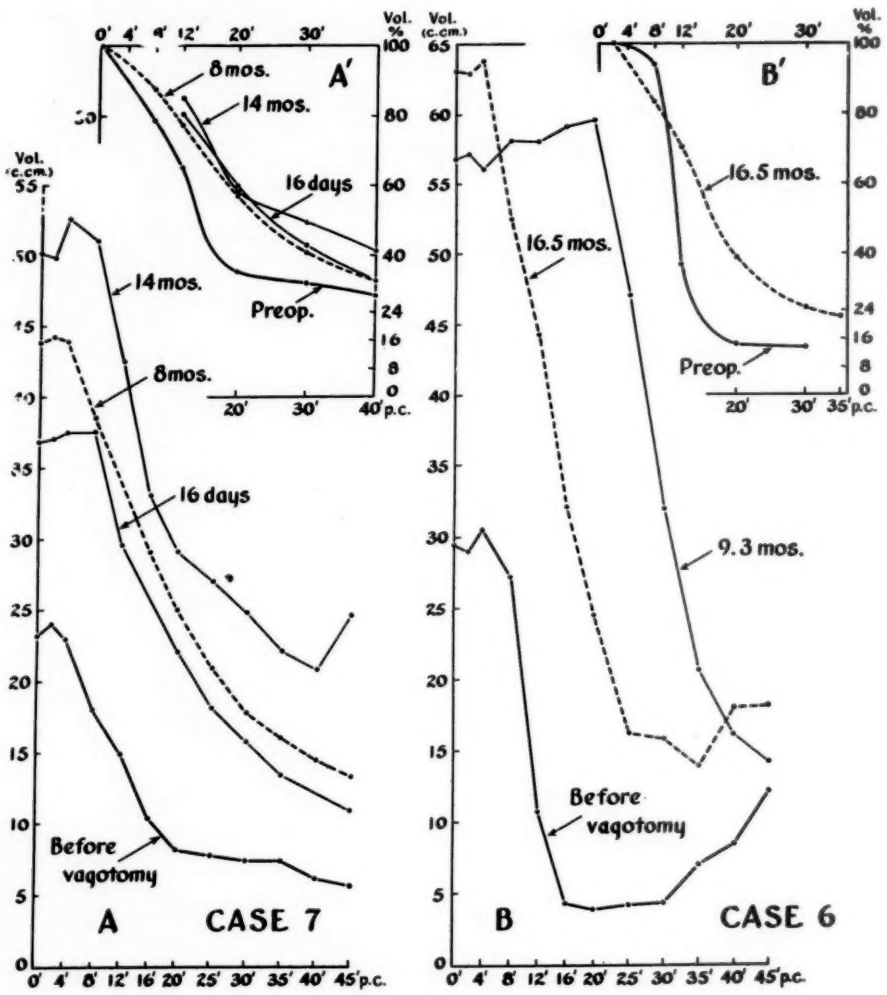


Fig. 5.

The next most instructive case is Case 6. Fig. 5, *B* illustrates the three series of cholecystograms, the lowest being preoperative, the next higher 9.3 months after a subdiaphragmatic vagotomy, the highest 16.5 months (or 6.5 months after a second vagotomy). Again the fasting volume of the gall bladder increases progressively in size. The first postoperative series reveals a marked

delay of twenty minutes before the gall bladder begins to empty, but then it empties at about the same rate as in the second postoperative series. When the per cent loss is compared (Fig. 5 *A'*), it is clear that in the second postoperative series evacuation of the gall bladder proceeds at a markedly slower rate than preoperatively.

### *Summary of Group B*

Case 1.—Preoperative Hollander test showed a rise in free HCl from 40° to 90°. Subdiaphragmatic vagotomy. Operation complete initially. At 8 days, no response to insulin; at 3 months, a rise of 18° in free acid (0° to 18°); at 1 year, 56° (0° to 56°); at 21 months, 60° (0° to 60°). At 3 months, rate of evacuation of gall bladder was markedly slower than preoperatively; at 21 months, rate was nearer the preoperative rate. Gallstone appeared.

Case 3.—Preoperative rise of free acid from 60° to 126°. Subdiaphragmatic vagotomy. Operation complete initially. At 8 days, no response to insulin; at 14 months, 24° (14° to 38°); at 22 months, 6° (16° to 22°). At 14 months, rate of evacuation markedly slower than preoperatively; at 22 months, rate is nearer the preoperative rate.

Case 4.—Preoperative rise of free acid not recorded. Maximum level 86°. Transthoracic vagotomy. Operation complete initially? At 8 days, a rise of only 12° (4° to 16°); at 8.5 months, 46° (10° to 56°); at 22 months, 66° (0° to 66°). Does this indicate regeneration of vagus nerves (cf. Case 1)? Moreover, this case is puzzling since at 22 months the gall bladder empties more slowly than preoperatively, in spite of the increasing response to the Hollander test.

Case 5.—Preoperative rise of free acid from 34° to 104°. Subdiaphragmatic vagotomy (and gastrojejunostomy). Operation complete initially. At 9 months, a rise of 8° (56° to 64°); at 15 months, 30° (40° to 70°). At 9 months, rate of evacuation somewhat slower; at 15 months, rate is faster than preoperatively (due to direct injection of meal into duodenum).

Case 6.—Preoperative rise of free acid from 12° to 90°. Subdiaphragmatic vagotomy (and gastrojejunostomy). Operation complete initially. At 3.3 months, no response to insulin; at 9.3 months, a rise of 24° (6° to 30°). At 9.3 months, rate of evacuation markedly slower; at 10 months a second operation (transthoracic vagotomy). 6.5 months later, no rise in free acid. Rate of evacuation unchanged.

Case 7.—Preoperative rise in free acid from 32° to 78°. Subdiaphragmatic vagotomy (and gastrojejunostomy). Operation complete. At 6 days, a rise of 10° (0° to 10°); at 6 months, no response (0° to 0°); at 13 months, 12° (0° to 12°). At 16 days, rate of evacuation markedly slower; at 8 months slightly faster than at 16 days; at 14 months, slightly slower than at 16 days.

## DISCUSSION

A scrutiny of Cases 7 and 6 would seem to leave room for no other conclusion than that in these patients, at least, the complete surgical interruption of the vagi fails to slow the emptying of the gall bladder beyond the level of return to normality. That is, the return to normal rate of emptying after vagotomy may be ascribed not to denervation of the gall bladder but to denervation of the stomach and to consequent improvement in the ulcer condition. This is supported by the work of Boyden and Berman[5] who demonstrated that the gall bladder empties faster in ulcer patients and postulated that it was due to the faster initial discharge of food from the stomach. Recently (in an as yet unpublished fluoroscopic study) Rigler and Boyden determined the amount of

time it took for at least 8 c.c. of egg yolk and barium to pass the pylorus. In 16 normal individuals an average of 6.3 minutes was required, but in 17 patients with duodenal ulcer the average time was only 3.7 minutes.*

In view of these facts, it is clear that in man, as contrasted with such laboratory animals as the cat, the vagi do not appreciably alter the rate of evacuation of the gall bladder after a fatty meal. This is in accord with the earlier observations of Boyden and Rigler[3]; namely, that faradic excitation of the stomach and duodenum in man, as contrasted with the cat, does not retard (through reflex action or otherwise) the discharge of bile from the gall bladder. These vagotomy experiments would thus seem to offer final proof that in man the evacuating mechanism of the biliary tract is primarily, if not exclusively, hormonal.

The second conclusion to be drawn from these vagotomy experiments is that complete severance of the vagi results in progressive enlargement of the gall bladder. No other circumstances are known to the writer in which such a relatively rapid increase in size of the viscus occurs in a given individual. In pregnancy, to be sure, distention due to stasis has been reported in 75 per cent of gravidas.[13] But Gerdes and Boyden,[7] in comparing the rate of emptying of five gall bladders during pregnancy and after parturition, found little change in the mean volume of the resting gall bladders. (During gestation it was 31.1 c.c. and 6 to 8 weeks afterward it was 27.4 c.c.) It is unlikely, therefore, that the consistent increase in volume in six ulcer patients from an average of 29.7 c.c. before vagotomy to an average of 61.3 c.c. after vagotomy could be due to anything but the effects of the operation—namely, the vagotomy. The inference is that the nerves which are responsible for the tonus rhythm of the gall bladder in the interdigestive periods have been severed. Since the gall bladder is then unable to resist the influx of liver bile as efficiently as before, its capacity must necessarily increase.

Most important is the question as to whether such enlargement of the gall bladder reduces its power of concentrating bile or leads to pathology of the organ. While, as yet, there is no answer to this question—and one usually associates gall bladder pathology with stasis—it may be pointed out that following vagotomy the gall bladder in two patients (Case 1 and Case 2) either developed a stone or failed to be visualized. Of course, these occurrences may be coincidental.

Another consequence of vagotomy would be the loss of those reflexes from gall bladder to gut, etc., which have been described by Schräger and Ivy.[15]

## CONCLUSIONS

From eleven out of thirteen male patients studied over a three-year period satisfactory series of cholecystograms were obtained both before and after vagotomy. These patients could be divided into two natural groups.

Group A, designated the negative control group, consisted of five patients in whom the vagotomy was incomplete as revealed by the Hollander insulin gastric

*Personal communication. For methods used, see their fluoroscopic study of the initial rate of emptying of the stomach in nullipara and primipara. (Proc. Soc. Exper. Biol. and Med. 56: 200, 1944.)

secretion test. With one exception, explainable on other grounds, these continued to exhibit the rapid evacuation of the gall bladder which is characteristic of peptic ulcer patients.

Group B consisted of six patients in whom the vagotomies were complete initially. In all of these the rate of emptying of the gall bladder was significantly retarded after vagotomy, but the rate was not less than that of normal individuals of corresponding sex and age. Accordingly, it is concluded that the vagi are not essential to evacuation of the human gall bladder (as contrasted with laboratory animals) and that emptying is regulated primarily through a hormonal mechanism.

However, in these six patients the fasting volume of the gall bladder was approximately doubled after vagotomy. This is believed due to the loss of vagus fibers which normally maintain the tonus of the gall bladder during interdigestive periods. This tonus failing, the viscus is unable to resist the influx of liver bile as efficiently as before, hence the gall bladder progressively enlarges. The question is raised as to whether such enlargement may lead to gall bladder pathology.

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# Editorial

## SIGNIFICANT SURGICAL MEETINGS

THE present scholastic year has seen a number of significant meetings affecting surgery. The meeting of the American College of Surgeons in San Francisco last October was significant from several points of view. First, the program was excellent; second, it provided further evidence of the fact that motion pictures and television can be important adjuncts in surgical teaching and training; third, at this meeting certain organizational changes within the College were further consummated—the Board of Governors of the College became a more significant group in the conduct of the affairs of the College, and finally, in probably the most significant of all its actions, the College began to formulate a concept which would strengthen the hands of the American Board of Surgery as the only accrediting board which should be recognized in this country, as far as general surgery is concerned. This action was further strengthened by resolutions adopted by the Regents of the College at the meeting of the Regents at Hot Springs, Virginia, on Dec. 3, 1951.

The American College of Surgeons has moved forward under the able leadership of its former Chairman of the Regents, Dr. Arthur W. Allen of Boston. It will continue to move forward under the leadership of Dr. Evarts A. Graham of St. Louis. These men have been dedicated to the best ideals of surgical practice; they have occupied pre-eminent positions in American surgery; they have not, and will not, fail to provide the necessary leadership which American surgery must have.

On Nov. 12, 1951, the Fifth Congress of the Pan-Pacific Surgical Association convened in Honolulu, Hawaii. The registration was twice as large as any of the four previous conferences of the Association. The registrants from forty-two states and eleven countries joined together to fulfill the purposes of the Association: (1) to exchange surgical ideas and methods, (2) to further the principles of hospital standardization and administration within the entire Pacific area, (3) to promote a spirit of good fellowship among the various races of the Pacific, and (4) to help to elevate the practice of surgery in the Pacific area.

The five-day scientific program with sessions in all divisions of surgery and related fields was well attended. Of particular interest were the informal breakfast meetings for the various specialty groups preceding the formal scientific sessions. The highlight of the Congress was the rededication of the Heiau Keaiwa (a medical temple of old Hawaii) conducted entirely by direct descendants of the highest chiefs of old Hawaii for the purpose of paying respect to the

healing art of that ancient group of islands. Honored guests were two doctors from each of the foreign countries represented at the Pan-Pacific Surgical Conference and one doctor from each of the States of the Union represented at the meeting. The interchange of ideas between surgeons of the United States and those of countries bordering on the Pacific Ocean will, without doubt, elevate surgical practice in the Pacific area.

The celebration at the Johns Hopkins Medical School of the centenary of the birth of William Stewart Halsted was associated with a group of meetings held under the auspices of the Centenary Committee. Great credit is due Dr. Emile Holman for having arranged this program. During the celebration the following societies held meetings: The Society of Clinical Surgery, the Halsted Club, the Society of University Surgeons. In addition, the Halsted Centenary Committee had a program of its own dealing with subjects peculiarly fitting for the occasion. The celebration was attended by Mr. D. H. Patey of London and Professor René Leriche of Paris. The papers presented at the meeting of the Society of University Surgeons were published in the August issue of SURGERY. The Halsted papers appear in this issue.

In December, the Southern Surgical Association met at Hot Springs, Virginia. The program was an excellent one; it provided the opportunity for a mutual interchange of ideas. Southern in its origin, it has, within more recent years, permitted participation and membership by men who are carrying on their work north of the traditional Mason-Dixon Line.

In April, the American Surgical Association, founded by Samuel Gross in 1880, held its meeting at the Greenbrier Hotel at White Sulphur Springs, West Virginia. An outstanding program was presented, both from the standpoint of papers dealing with purely experimental subjects and from the standpoint of clinical investigation. Over the many years that this Society has functioned, it has continued to uphold the highest traditions of surgical practice. The roster of its past membership includes the names of those who have made the most significant contributions to American Surgery.

These are but a few of the many worth-while meetings of surgical societies and associations during the past few months. To name them all would be a task. The programs in nearly all instances have been good.

The future of American surgical science and practice will be assured as long as those who are imbued with the finest traditions of surgical scholarship continue to control these and other associations and societies which have in the past had such an important effect in molding the future of surgery in this country. There should be no compromise with those who would lower the standards of surgery; there should be no compromise with those who would lower the standards of hospital practice; there should be no compromise with those who violate decent morals and ethics in surgery.

The recent action taken by the Regents of the American College of Surgeons regarding unethical practices, such as fee-splitting, should be heartening to all surgeons who are deeply interested in maintaining the finest traditions of medicine.

The mandates for the future are clear. We must continue to strengthen the triad upon which competency must be based: (1) the care we give our patients, (2) the teaching and training of young men, (3) the contributions which are made toward a better understanding of the problems of surgery. In addition to these aims, we must rededicate ourselves to eliminating, by every means possible, the practices of fee-splitting in surgery, ghost surgery, unnecessary operations, and over-charging patients. The outstanding surgical organizations in this country must take the necessary leadership in maintaining the highest ideals of surgical practice. It is only by doing this that the practice of surgery in this country can continue to make progress.

*—I. S. Ravdin*